volume 2

CLINICAL PROPEDEUTICS AND MEDICAL SEMIOLOGY

RAIMUNDO LLANIO NAVARRO
GABRIEL PERDOMO GONZÁLEz

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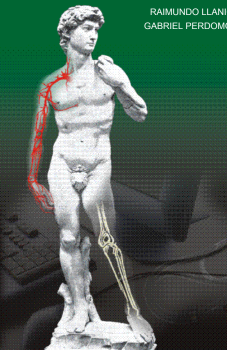

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volume

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To my wife Flora Villate, for her help in preparing the book, for her moral support and, above all, for her understanding, patience and sacrifice during the thousands of hours that have delayed the completion of this work.

To the students and doctors, past, present and future, owners of the future of medicine, to whom I have dedicated a large part of my life.

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NERVE SYSTEM0. CLINICAL HISTORY AND CHANGES IN THE GENERAL EXAMINATION

50

MEDICAL RECORD

The examination of the nervous system, like that of the other systems, begins with the preparation of the patient's medical history; To do this we will also use the same examination procedures that we have indicated for the rest of the body's systems, such as:

the interrogation as a technique to obtain subjective symptoms, the general physical examination to collect objective symptoms and instrumental (complementary) examinations.

INTERROGATION

The old aphorism that: in the nervous system the examination is everything and the interrogation nothing, must be discarded. It is true that the physical examination is capable of providing rich information about the neurological patient and that much of this is of extraordinary precision, particularly for topographic diagnosis (that is, to be able to indicate at what level of the neuroaxis a certain lesion is located); But the interrogation is also essential and there are many neurological conditions in which the physical examination is totally negative and only the interrogation allows the diagnosis. The most important example is the most frequent forms of epilepsy.

The interrogation carried out by a well-trained doctor allows us to suspect most of the conditions of this system, since according to the different subjective symptoms that accompany or precede a certain clinical condition, the various causes that may originate it are raised. For example: a hemiplegia (paralysis of half or one side of the body) immediately makes one think of the nervous system and the site of the injury; the fact that it has started suddenly makes one think of a hemorrhagic or embolic accident as the cause; while the same picture, if it has been established slowly and insidiously, progressing from one moment to the next, from one hour to another, until the described lesion is formed, will suggest a thrombosis as its cause. If the condition were to establish itself slowly and progressively, but over the course of days and weeks, rather than in a vascular-encephalic type disease, we would think of an intracranial expansive lesion (tumor, for example).

The interrogation is carried out during the medical interview, which involves the relationship between two people, mainly through language.

purely verbal and also extraverbal (facial expression, gestures, periods of silence in the interview, etc.).
During the interrogation, the doctor observes the patient's personality, state of mind and cultural level, which allows him, according to these characteristics, to individualize the form of the interrogation. Interrogation conceived in this way is an art, and can only be developed with continued and careful practice.

It not infrequently happens to the student or young doctor that his teacher or a more trained doctor obtains from the patient an important piece of information from the interrogation, sometimes a true key to the diagnosis; This occurs despite the student having shown the greatest interest and having spent a lot of time with the patient. Generally the reaction is to blame the patient, attribute the fact to their low cultural or intellectual level, to the image of authority that the teacher represents, etc. But it is not what really happens, but rather the teacher masters the art of interrogation and knows how, when and in what way to ask a certain question and, furthermore, knows how to adapt the interrogation to any type of patient, that is, to an observant patient or to one who is distracted or imprecise; to one intelligent and with a high cultural level or to one more clumsy and with less culture.

The possibilities of interrogation should not be considered finished in the first interview. In a second encounter, the patient is generally calmer or more confident with the observer, and then important data can be obtained that were not obtained in the initial contact.

The time for the interrogation varies greatly, depending on the doctor's expertise and the type of condition the patient suffers from.

An interrogation is, as we already said, a communication between two people through a code: language. Both the patient and the doctor frequently have an incorrect tendency during the interview:

abbreviate descriptions using short qualifiers or technical terms. This habit can produce a true withholding of information. Let's look at an example:

A patient complains of having dizziness. The interrogator accepts and transcribes the term literally, or replaces it with vertigo, which is essentially the same, but which gives a greater impression of certainty. But it happens that people have the most varied conceptions of the term dizziness. For a patient, dizziness will actually be a spinning or rotating sensation of himself or things around him. In this case it will really be vertigo; But for quite a few dizziness is cloudy vision or other visual sensations, or a feeling of fainting, or other undefined sensations of instability, etc.

For this reason, the interrogator should not accept terms of this type, but rather get the patient to describe his subjective sensations, and in most cases transcribe this description almost verbatim so that everyone (and not just him) has access to the primary data.

Questioning the patient is often insufficient; Not infrequently, family members and cohabitants must be questioned so that the information acquires true coherence. There are even cases (seizures, or the beginning of a stroke) in which the patient cannot provide a single piece of information, and it is the family members or other people who witnessed the event who can do so.

The main reason why the patient went to the doctor must also be specified. Sometimes this motivation constitutes a symptom of great value, but on other occasions the symptom is less important, despite which it is essential for the patient. In all cases, the doctor is obliged to know the reason for this importance. (Sometimes it is not the symptom itself, but what the patient thinks about that symptom, that motivates him to consult.)

It must be kept in mind that a point of great importance is that the history of the disease is part of the patient's own life. It is not something that can or should be artificially isolated from aspects concerning work or family life, and it must always be placed in this real social context.

Likewise, it is necessary in the interrogation and through it, to explore the patient's intellectual state. It should not be considered that the neurological examination can dispense with the mental status examination and vice versa. The nervous system is but one and its examination must be comprehensive if it is to be complete. This examination will allow us to observe the repercussions that organic lesions produce on the mental state, and also, through the analysis of the psychological symptoms and their integration into a syndrome, to consider the multiple processes that can cause them.

The history of the disease must be obtained in chronological order and written in this form, that is, the chronopathogram must be

For each symptom, the following scheme or order must be followed:

1, Date of appearance of the symptom.

2. Location, intensity and extension.

3. Factors that accentuate or diminish it.

4. Evolution of the symptom (constant improvement or worsening or crisis, changes over time, etc.).

5. Effects of previous treatments.

6. Associated symptoms.

7. Result of previous investigations (if any).

PHYSICAL EXAMINATION

The special and particular methodology of the physical examination of the nervous system was already explained in Chapter 14, Section I.

In this chapter, and in the subsequent ones dedicated to the physical examination, we will study the alterations that can be found, in each of its parts, which completes the exploratory technique and allows us to evaluate the physical signs found, to establish neurological syndromes and presumptive diagnoses.

INSTRUMENTAL EXAMS

The instrumental examination will always be guided by the data obtained from the interrogation and the physical examination, and will be aimed at specifying, above all, the extent of the process and its etiology.

The different procedures that make up this exam will be indicated in Chapter 53 corresponding to complementary exams.

ALTERATIONS IN THE GENERAL PHYSICAL EXAM

PATHOLOGICAL FACIES

The normal appearance of the face depends on the integrity of the muscles and nerves of each of the halves of the face, as well as the state of the structures of the central nervous system that govern them, so alterations in any of these structures (peripheral or central) cause alterations in the facies.

Facies alterations may be located on one side, which will occur more often when peripheral structures are affected; while alterations of the higher structures will more frequently produce global facies disorders.

In the case of localized alterations, it is possible by simple inspection to specify the affected cranial nerve; and in the case of non-localized alterations, suspect the diagnosis of the disease, also by simply observing the face.

Hence the importance that the study of facies has in semiology.

semiography

The facies produced by alterations in one half of the face will be studied within the alterations of the cranial nerves. Here we will limit our study to the facies in which the entire face is affected and to the facies of capsular hemiplegia, which is practically half of the face.

Facies of patient with capsular hemiplegia during coma

Here the face is symmetrical, although it appears wider on the paralyzed side. The cheek on the paralyzed side moves with each respiratory movement as if it were an inert veil, lowering on inhalation and rising on expiration more than that on the healthy side, which constitutes the sign of the pipe smoker. There may be anisocoria (unequality of pupil size) with mydriasis (increase in pupil size) on the side of the injury, that is, on the opposite side to the paralyzed one. The nasolabial fold may be marked on the healthy side. There may be conjugate deviation of the head and eyes. When this occurs, the head and eyes deviate to the opposite side of the paralyzed person, and that is why it is said that in these cases the patient "looks towards his injury." The facies described corresponds to a hemiplegia produced by injury to the internal capsule (capsular hemiplegia), where the corticonuclear bundle, a component of the pyramidal bundle at the level of the internal capsule, is injured. In rarer cases, this facies can also be caused by a cortico-subcortical pyramidal lesion. In others, the conjugate deviation of the head and eyes is not present in the capsule lesion.

Facies of the pseudobulbar patient

It is generally an old man, the facies is expressionless, lifeless. Sometimes saliva comes out of the corner of the mouth, which expresses poor muscle control. The patient presents attacks of unmotivated crying or laughter, and considerable difficulty in articulating speech (dyslalia), as well as swallowing and phonation. The march is in small steps. This disorder is accompanied by alterations in the highest functions of the nervous system (cognitive, volitional and affective functions) that give the patient the general appearance that the common people know as "old man."

This alteration is related to bilateral brain lesions of the corticobulbar pathways, of atheroscleroic or senile origin. Its name corresponds to an attempt by the classics to differentiate it clinically from bulbar palsy, an entity of another nature that affects the nuclei of the cranial nerves located in the medulla oblongata (VII, X, XII) and that also presents disorders of speech, phonation and swallowing.

As mentioned, in this last disease the lesions exclusively affect the nuclei of the cranial nerves, while in the pseudobulbar patient the lesions are diffuse and central.

Facies of Parkinson's disease and parkinsonian syndromes

The facies is expressionless, statue-like; the face is flaccid, mimicry is diminished or abolished. Above the eyes, the forehead often appears wrinkled due to contraction of the frontal and blinking is reduced (see Fig. 24.19, volume 1).

This facies frequently has a peculiar shine, expression of seborrhea or increased skin fat.

The mouth is half open (sometimes there may be trembling of the lips or tongue) and saliva flows through it, which sometimes hangs on the lip like the "drooling of infants." This sialorrhea is related to an increase in secretion and a decrease in the automatic swallowing movement. Seborrhea and sialorrhea are more intense in postencephalitic parkinsonian syndrome than in Parkinson's disease.

Added to the above is a typical tremor of the arms and hands, a rest tremor, which in the thumb and index fingers resembles the movement of counting coins; as well as a characteristic attitude and gait.

Parkinson's disease is caused by degenerative lesions of the substantia nigra (locus niger) of the mesencephalic tegmentum and the striatum of the telencephalic gray nuclei mainly, although it can also affect the cerebral cortex and the intermediolateral horn of the dorsal cord. In this disease

They damage the pathways of the extrapyramidal system.

Parkinsonian syndromes have a similar clinical picture, but their cause is known (encephalitis, atherosclerosis, toxins, medications).

Facies of myasthenia gravis

Myasthenia gravis (Fig. 50.1) is an autoimmune disease with a genetic predisposition caused by the destruction of acetylcholine (Aco) receptors on the presynaptic membrane of the nmotor plate, caused by complement-fixing antibodies. It is characterized by a lack of tolerance to normal muscular activity, that is, exaggerated fatigue and rapid exhaustion after exercise. There is a marked decrease in muscle strength, which recovers somewhat after rest.

The muscles of the face are frequently affected, particularly the levator palpebrae superioris, the oculomotor muscles and the muscles innervated by the facial muscle.

Typical of the myasthenic facies is eyelid ptosis, generally incomplete, and its general asthenic appearance, as if tired or fatigued.

Eyelid ptosis is commonly bilateral, but can be unilateral, and is often more marked than side. This ptosis sometimes causes a characteristic position of the patient, who tilts his head backwards to be able to see through the limited eyelid opening that remains.

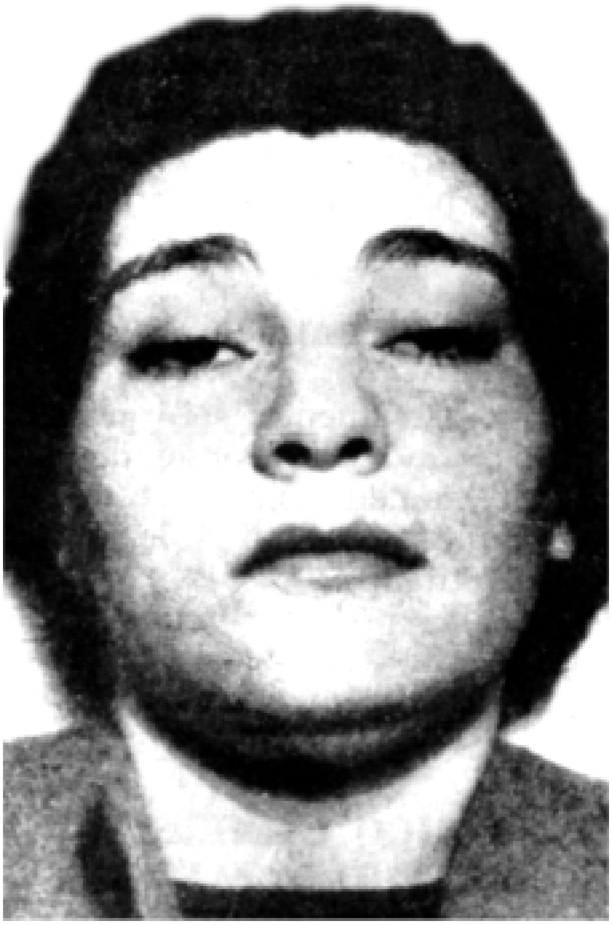


Fig. 50.1 Hutchinson's facies in myasthenia gravis. Astronomer's look.

Eyelid muscle weakness can be demonstrated by stress testing (Fig. 50.2).

Mimicry is diminished; The patient finds it difficult to whistle or smile, and after talking for a while he may become hoarse or dysphonic, all in relation to muscle weakness and fatigue.

Tetanic facies (or sardonic laughter) and strychnine poisoning

Fortunately, it is rare, because in our country tetanus has been practically eradicated by systematic vaccination, and strychnine is currently a drug of very little use.

In this facies there is exaggerated hypertonia with contracture of all the muscles of the face. The wrinkles on the forehead are very marked. The wings of the nose and eyebrows are raised. The palpebral slits are narrowed, the palpebral commissures are drawn towards

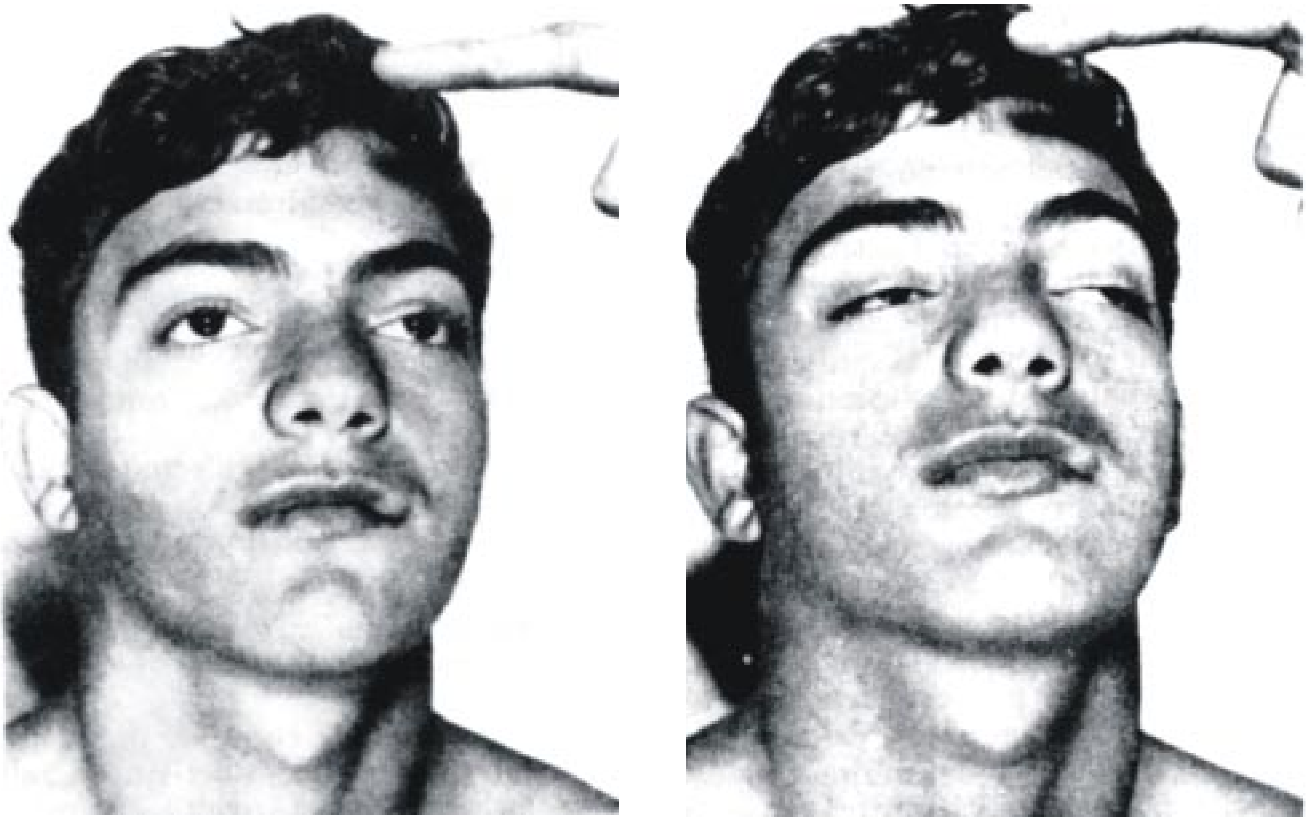


Fig. 50.2 Upper eyelid drooping test in myasthenia: a, the patient is asked to look up (towards the finger); b, after 5 minutes you notice that the upper eyelid droops.

outside. The corners of the mouth are also drawn upward and outward, leaving the teeth more or less exposed. As a whole, facies in its upper half shows an expression of pain and in the lower half a kind of permanent laughter, which is why the whole has been called facies of sardonic laughter or cynical spasm (see Fig. 24.20, volume 1).

In addition to the facies mentioned, we must always remember that the facies can tell us a lot about the patient's psyche and that it frequently allows us to detect anxious or depressive states.

STANDING ATTITUDE

In the face of an abnormal standing attitude or the impossibility of acquiring the normal one, it is necessary to ascertain whether the cause of it depends on an alteration of the locomotor system or the nervous system. With that reservation made, we will now deal only with the attitudes determined by alteration of the latter.

semiography

Parkinson's

In this state, the patient tilts his head forward, bringing his beard closer to his chest, and also flexes his trunk forward. It is rigid and may present tremor at rest (see Fig. 24.6, volume 1).

Capsular hemiplegia with spasticity

Asymmetry of the face due to paralysis of the lower portion of the facial nerve on the paralyzed side (sparing the forehead and eyelid). The paralyzed upper limb is flexed; It presents in the hand, the fingers all flexed on the palm and imprisoning the thumb under them; The arm and forearm are tightly pressed against the thorax, the forearm flexed over the arm.

The paralyzed lower limb is extended; the foot is plantar flexed, with slight adduction and internal rotation; the leg and thigh in forced extension. All of this gives greater length to this limb compared to the other lower extremity, so the patient tilts the trunk slightly on the healthy side, and to do so elevates the hip on the diseased side (see Fig. 24.5, volume 1).

Advanced ataxia

Patients who suffer from it have difficulties maintaining balance and to achieve this they remain erect, with their feet wide apart, in order to increase the body's support base; the body is oscillating and

Sometimes there is a tendency to fall forward (anteropulsion), backwards (retropulsion) or to one side (lateropulsion).

Myopathies or muscular dystrophies

A wide separation of the feet, accentuated lordosis and prominent abdomen are observed, giving rise to the classic tenor posture (see Fig. 24.7, volume 1).

Spastic paraplegia of the extension type

The patients take a characteristic attitude, their lower limbs touch each other at the knees, keeping their feet as if glued to the floor and they can barely lift the tips of their feet.

Korea

In chorea (from the Greek koeia: dance) the patient constantly has all his muscles in action with involuntary, rapid and sudden movements that affect one or more muscles of the trunk, face and extremities.

Unnecessary gesticulations appear on his face, he constantly moves his arms, wrings his hands and sometimes has one shoulder lower than the other (due to hypotonia of the trapezius muscle). In the hand of the choreic, a characteristic attitude is observed: the radiocarpal joint in flexion with the fingers in hyperextension at the level of the metacarpophalangeal joints and the thumb in abduction. When the patient attempts to raise the upper limbs to a vertical position, the palms are placed facing each other and the pronation of the forearm is sometimes so pronounced that the thumb projects forward.

ATTITUDE IN THE BED

semiography

Hemiplegia during coma

Generally in this state the hemiplegia is flaccid and is characterized by a special attitude that we will describe:

The body, on the bed, appears as if it were drawn toward the paralyzed side, because there is an apparent widening of the side of the paralyzed body. This is because the paralyzed muscles have lost their tone and the force of gravity, acting on them, depresses them vertically and they appear widened laterally. The thigh, instead of being a truncated cone, as normally observed, is now like an ovoid, flattened vertically. The external rotation that occurs at the level of the coxofemoral joint on the paralyzed side makes that limb appear even wider.

Opisthotonos, emprostotonos, pleurototonos

These attitudes are observed in patients with states of great muscle hypertonia: tetanus, strychnine poisoning, etc. They have already been described in Chapter 24, volume 1.

Orthotones

It comes from the Greek orthós: straight; tones: tension.

In cases of orthotonus, all the muscles are in contracture, both the flexors and the extensors, the body forms a straight and rigid block that can be lifted in one piece by the heels or the back of the neck (fig. 50.3).

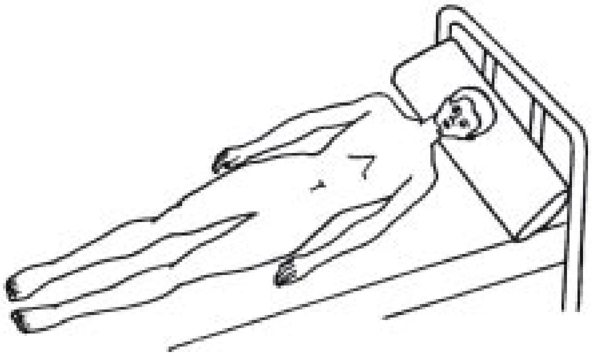


Fig. 50.3 Attitude in orthotones in strychnine and tetanus poisoning.

shotgun trigger attitude

It has already been described in Chapter 24, volume 1.

MARCH

Many disorders and diseases can affect gait.

We will deal here only with gait disturbances that are related to diseases of the nervous system.

Semiography and semidiagnosis

The study of gait is of great importance because it initially allows a semiological impression that the rest of the examination will confirm or discard. In this sense, it is advisable to proceed according to the following stages, where we will specify the details they provide, in a general way, and then address the description of the most important steps in the nervous system.

1. There are patients who are confined to bed and ambulation is impossible for them.
2. If they are able to walk, it is worth observing how they sit in bed, since by studying the gait of myopathies we will see the difficulties these patients have in getting up.

3. You have to see how they stay upright; if they spread their legs too far to maintain balance (a fact seen in patients with the vestibular pathways), or if they hesitate when standing, having to balance towards the four cardinal points to avoid falling (seen in patients affected by lesions of the posterior cordonal systems or the cerebellar pathways and centers.

4. They will have to be made to walk with their eyes open. A few will do it correctly; others, however, will show a series of alterations of the highest interest

semiological, for example:

a) In some, only one lower limb is useful, the other presents alterations and difficulties in walking (they are hemiplegic).
b) Others will have difficulties walking due to disorders in both lower limbs (the case of paraparetics).

c) Some will not be able to walk in a straight line and will do so in a zigzag (they are the cerebellars).
d) There will be patients who, in order to walk, must raise their leg and foot, which is fallen (paralyzed), so as not to collide with the ground; They are patients with paralysis of the external sciaticopopliteal nerve and have steppe.

e) Once the patient is standing, it is necessary to see if he is able to stand with his upper limbs next to his body, his eyes closed, and his feet together. If it has a tendency to fall, we say that the Romberg maneuver (which is what this test is called) is positive. This is true in patients with vestibular lesions. There is a kind of dogma (although in medicine there are no dogmas) according to which cerebellars do not have Romberg."

f) Sometimes the patient is made to walk, with his eyes closed (or blindfolded) back and forth, marking with chalk on the floor the departure and arrival from the various points, which together will give the figure of a star. This is known as star gait and is found in vestibular patients (see Fig. 15.21, volume 1).

We will now describe the most frequent types of functioning in the nervous system.

Paretic gait

Corresponds to: lesions of the peripheral nerves (polyneuropathies); isolated lesions of the anterior horn motor neurons (the main of which is poliomyelitis, disease eradicated in Cuba since 1970 by the health work developed in our country) and lesions of the pyramidal tract in the spinal cord.

In the case of lesions of the spinal cord, the gait is never pure paretic, but rather paretospastic, because the spasticity typical of lesions of the pyramidal tract is added to the paresis. Acute spinal cord injuries, which are not accompanied by spasticity, almost never allow walking.

Therefore, in practice, we observe paretic gait in polyneuritis, or better, in polyneuropathies.

Paretic gait is difficult; The patient sometimes drags his feet and looks at the ground to help with vision, especially if he suffers from polyneuritis that affects fibers that conduct proprioceptive sensitivity. In many cases the dorsiflexion of the foot is very difficult and when the leg is raised, the tip of the foot is tilted downwards, as if the foot were hanging from the leg; then, in order to walk without dragging the tip of the foot on the ground, the patient raises the thigh and leg higher than normal, and when he is going to let it fall the first thing that touches the ground is the tip and the external edge of the foot, producing a characteristic noise. This gait has been called steppe, because it recalls the march of the steppe horses (see fig. 24.8, volume 1).

spastic gait

The most typical is that seen in capsular hemiplegia with contractures. The patient in the standing position adopts the attitude described, and as we said there he faces the problem that his lower extremity paralyzed in extension is longer than the healthy one, and due to spasticity he cannot dorsally flex the foot on the leg, nor the leg on the thigh, for all this it is very difficult (sometimes impossible) for him to separate the tip of the foot from the ground, and to walk he then performs a circular movement, similar to that of a scythe. to mow, describing a semicircle with the diseased foot that has the healthy foot as its center, that is, the diseased foot rotates around the healthy one, resting mainly on the toe and external edge of the affected foot, these parts of the footwear that wear more intensely; In addition, a characteristic noise is produced that allows the gear to be identified.

This type of gait is known by the names: scythe, helical, reaper or Todd gait (see fig. 24.8, volume 1).

ataxic gait

This type of gait is found in patients who have altered coordination of movements, although in them there is no alteration of muscle tone, paresis or paralysis. In it we distinguish three main types:

1. Tabetic march. Tabes dorsalis is a condition caused by syphilis in its tertiary period and characterized by a lesion of the posterior roots of the spinal cord and the posterior cord. In our times, with the greater control and cure of syphilis, this march is rarely observed. A similar pattern can be observed in some forms of polyneuritis. It is called polyneuritic pseudotabes. Tabetic gait is characterized by the patient looking at their own gait to find out about the position of the limbs, data that does not reach the corresponding centers due to injury to the posterior cord (the eyes are the tabetic's crutches). The gait is also performed with exaggerated movements when raising the leg, which is then dropped abruptly to the ground, touching the ground first with the heel (the tabetic talonea) (see Fig. 24.8, volume 1).

2. Cerebellar gait (see Fig. 24.8, volume 1). When standing, the cerebellar wobbles abnormally (static ataxia). Therefore, it opens its legs to increase its base of support. During walking, these oscillations are greater (dynamic ataxia), the separation of the legs is also maintained, the gaze is directed towards the ground and the gait is characterized by being zigzag, like that of a drunk person. That is why it is also called a drunken walk." There is a tendency to fall to one side (lateropulsion), forward (anteropulsion), or backwards (retropulsion). The most frequent is lateropulsion: it can be towards one side or the other, if the lesion is diffuse or bilateral. If the cerebellar lesion is unilateral, lateropulsion occurs towards the same side where it sits. Lateropulsion can also be seen in the syndrome vestibular. Incoordination when walking is evidenced by exaggerated movements during walking (the patient raises the knee exaggeratedly and the leg goes forward more than necessary).

3. Gait of the patient with vestibular syndrome (see below cranial nerve VII, in Chapter 52).

Walking in small steps

It is mainly observed in patients who have alterations of the striatum (Parkinson's disease) and in pseudobulbar patients.

It is characterized by the patient taking very small steps, without ever passing one foot in front of the other, that is, when the left foot advances forward, for example, it reaches a distance, but when the right foot advances immediately, it never passes further in front of where the left foot is and perhaps does not reach it. Sometimes the small steps are slow, which is called bradybasia, other times they are very fast together. In Parkinson's disease, moreover, the patient walks leaning forward, giving the impression that he is " running after his center of gravity."

Digitigrade gait

It is a variety of pareticospastic gait. It is observed in spastic paralysis of the two lower extremities, that is, in the so-called spastic paraparesis. As both lower extremities are paralyzed in extension, the patient is practically standing on the tips of his toes and cannot perform dorsiflexion movements of the foot on the leg at the level of the tibiotarsal joint, nor can he flex the leg on the thigh at the level of the knee joint or the thigh on the pelvis at the level of the coxofemoral joint. For all these reasons, to walk you have to take small steps, dragging the tips and inner edges of the feet and wearing the shoes markedly in those regions (see fig. 24. 8, volume 1).

Scissors walking

It is observed mainly in congenital cerebral diplegias. They are people who maintain paraplegia with plantar flexion of the feet, with them being separated at ground level, but the legs tend to get closer to each other, contacting at the level of the knees, thus the two legs form a triangle with the upper vertex with the ground; The legs are slightly bent over the thighs and the thighs over the trunk, so that the standing patient appears to be sitting or squatting.

To walk, these patients alternately cross one leg in front of the other, in an "X" shape, touching both at the knees and wearing their clothes at that level. As is easily understood, this is also a type of pareticospastic gait (see Fig. 24.8, volume 1).

Progress of primary myopathies

In progressive muscular dystrophy there is marked muscle atrophy due to damage to the muscle itself, of genetic origin.

The muscles most affected are those that straighten the trunk, those of the pelvis and those of the thigh. It is observed mainly in children in second childhood. The march resembles that of people who want to be important; belly forward, chest raised, great increase in lumbar saddle, with movement of the thighs reminiscent of duck walking ("waddling" gait). As a whole, this march has also been called dandy.

clawn march in korea

The choreic patient advances with oscillating movements of the trunk, giving the impression with each step that he is going to fall, which, together with the sudden and disordered movements of the arms and trunk, recalls the gait of the claw.

toad march

It occurs in the advanced stages of myopathies (progressive muscular dystrophy) when the patient can no longer stand, but can do so by resting the fingers and toes on the ground and squatting (Fig. 50.4).

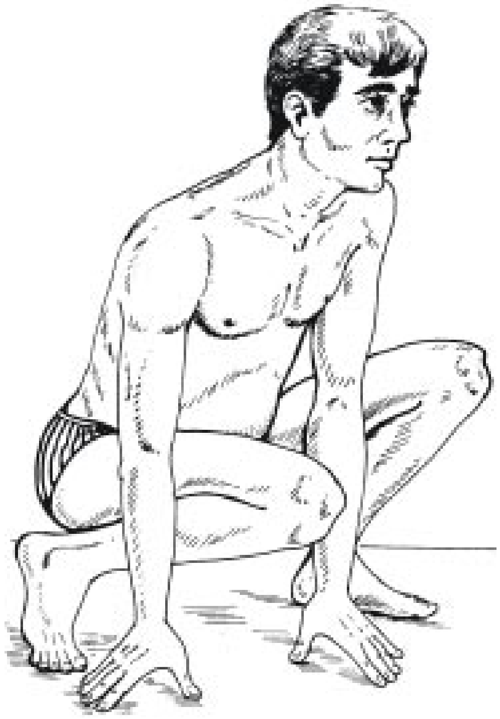


Fig. 50.4 Toad gait in advanced myopathies.

NERVOUS SYSTEM. ALTERATIONS IN YOUR PARTICULAR PHYSICAL EXAMINATION

51

TROPHISM

CONCEPT

Trophism is called the optimal conditions of nutrition, development, renewal and life of certain tissues (such as the skin, the subcutaneous adipose tissue, muscle and bone tissue) and joints. Trophism is regulated by the nervous system.

As mentioned in Chapter 14, volume 1, the exploration of trophism is carried out throughout the physical examination, mainly of the skin and the osteomyoarticular system. But in the particular physical examination of the nervous system, before or after having performed the examination of the cranial nerves, we proceed to inspect the patient to observe the symmetry of his limbs and the two halves of the trunk. If one of its members (or part of them) is diminished in relation to the other; or if both limbs are diminished in relation to the muscular development observed in the rest of the body; or if a part of the trunk, neck or both is decreased, compared to the opposite side. If there is any difference, it is suspected that this area is atrophied, which we will verify later by other exploratory means.

SEMIOGENESIS OR PHYSIOPATHOLOGY

The skin is closely related to the nervous system not only due to its innervation, but also due to an embryological affinity, since both come from the ectoderm. Vilanova and Dulanto affirm that the skin is the external part of the nervous system. The responsibility for cutaneous eutrophy falls mainly on the afferent vegetative and sensory fibers; in the latter, because by transmitting sensitive and sensory impressions that inform of the presence of any harmful agent, they motivate a defensive response; in vegetative fibers, for its governing role in vasomotricity and glandular functioning and its direct influence on the skin.

Muscle trophism is under the control of the peripheral motor neuron, located in the anterior horns of the spinal cord, and its equivalent the somatic motor nuclei of gray matter, which are spread throughout the height of the neuroaxis and which serve as a starting point for the motor fibers of the cranial nerves. Trophic stimuli are carried by motor nerves to the muscle. In this way, any injury to the nerve or the cell from which they originate will manifest due to the atrophy of a muscle or the part of the muscle that said nerve or motor fibers innervate.

After section of a motor nerve, there is loss of sarcoplasm and a reduction in the caliber of denervated muscle fibers. After two months, there has been a reduction in fiber caliber that can reach up to 70%, and the muscle cells are greatly reduced in size. Subsequently, an increase in connective tissue occurs. All of these changes, which are progressive in nature, are known as denervation atrophy.

There are diseases of the muscle itself that can affect its trophic state. These conditions are of genetic origin and are assumed to be an enzymatic defect or muscle protein structure defect. Other muscle atrophies are of inflammatory, metabolic, nutritional or endocrine origin.

SEMIOTECHNICS

1. Inspection of the muscular regions, which must be completed by studying the condition of the skin, bones and joints, which are also affected by trophic disorders.

2. Palpation of the regions being explored and study of the degree of consistency of the muscles, their volume - in cases where they can be covered with the hands - and their elasticity. The certainty of the existence of muscular atrophy, in cases of doubt, will be obtained by measuring the muscular areas and comparing them with the symmetrical ones on the opposite side.
   To do this, the measurements must be taken in equal areas of the two limbs, so it is always advisable to look for a bone reference point, from which the distance is determined, parallel to the long axis of the limb, in which the circumference measurement will be made. For example, in atrophy of the calf muscles, a distance is measured from the lower edge of the kneecap, along the limb, and then at that distance the circumference of the leg is measured. Then we go to the other leg and from the lower edge of the kneecap we measure the same distance as in the other leg and it is at that distance where we determine the circumference of the other limb, to compare them. Is-

   These measurements should be made with a cloth measuring tape that is very malleable, so that it can be applied without difficulty to the surface of the body to be measured. Metallic tapes are not suitable for this, as they do not adapt exactly to the circumference of a member, for example.

SEMIOGRAPHY AND SEMIODIAGNOSTICS

Trophic or nutritional disorders constitute an important part of the symptoms of numerous neurological processes and manifest themselves at the level of the skin, nails, subcutaneous tissue, muscles, bones and joints.

Classification

Trophic disorders can be classified into:

- Skin and nail alterations.
- Joint alterations.
- Bone atrophy or osteoporosis.
- Alterations of myotrophism.

Skin and nail changes

1. Shiny and smooth skin:
   a) Hyperhidrosis (excessive sweating).
   b) Anhidrosis or hypohidrosis (absence or decrease of sweating).
   c) Whitish and choreous skin.
2. Cyanosis or paleness.
3. Hypertrichosis (excess hair).
4. Hypotrichosis (lack of hair).
5. Edenma of the skin.
6. Rough and brittle nails.
7. Trophic ulcers or poor perforating ulcers with slow healing.

Joint disorders

They appear in the tabes. The so-called Charcot joints are painless despite the marked destruction of cartilage, ligaments and articular surfaces. In these cases there are intra-articular bone fragments, and an excess of synovial fluid fills the cavity and pathologically increases joint mobility. Large joints, such as the knee, hip, and ankle, are often the most affected.

Bone atrophy or osteoporosis

It accompanies paralysis and lack of joint movement.

Semiological alterations typical of myotrophism

The following types of muscle disorders can be found:

1. Atrophies (from the Greek atrophia: lack of nutrition). They constitute the most frequent alterations; there is an evident decrease in the volume of muscle masses.
2. Hypotrophies. They tend to be the first step on the way to muscle atrophy or, on the contrary, be diffuse, as seen in patients confined to bed for a long time.

3. Pseudohypertrophies. In the inspection of muscle 0, or rather, muscle masses, they appear as if they were " increased in volume." Palpation sometimes reveals increased consistency (wooden muscles", as seen in some myotonias) or, on the contrary, a lower muscle tone, as seen in myopathies (diseases of muscular origin and with a genetic nature, such as the pseudohypertrophic variety of Duchenne).

4. True hypertrophy. In this type, certain forms characteristic of some athletes must be discarded (due to the effect of methodical and prolonged professional exercise).
   In the histological examination, some hypertrophic fibers can be observed, as occurs in certain myopathies.

From a semiological point of view, muscular trophic disorders can be classified according to various etiologies: neurogenic, myopathic, encephalic and other types.

First of all, we must distinguish true muscle atrophies from those called atrophies due to disuse. The latter are nothing more than the decrease in volume of a muscle or group of muscles of an extremity or even half of the body, which occurs when that muscle for some reason is not exercised, it does not perform the function it is supposed to do. For example, it is common for people who have an injury to their brain in the motor area to develop paralysis, the extent of which varies, in the area on the opposite side of the body, to that on which their brain injury occurs. As a consequence of the paralysis thus produced, that person cannot move one leg or even half of the body, and as a consequence of the lack of use of that limb or those limbs, the muscles of the same are reduced in size in relation to those of the healthy side. This is not actually muscle atrophy in the true sense of the word, since the first motor neuron or pyramidal neuron has no function on muscle trophism.

Neurogenic muscle atrophies due to damage to the neuron of the anterior horn of the spinal cord are accompanied by paralysis of the affected muscles, which are markedly hypotonic and may present fasciculations (sudden contractions of parts of the muscle, visible through the skin). They generally predominate in the distal muscles.

Muscular atrophies due to muscle disease (myopathic atrophies) begin in childhood or adolescence, are genetic in nature, show a predominance of proximal localization (root of the limbs) and on the face, coincide with loss of strength and are accompanied by enzymatic alterations (transaminase). glutamicOxalacetic, aldolase, phosphocreatine kinase). This group includes ocular myopathies, which can extend to other muscles, do not have fasciculations, there may be no hypotonia and they predominate in the proximal muscles (pelvic and scapular girdles).

Electromyography is essential for diagnosing the cause of muscle atrophies; it checks the degeneration reaction in the neurogenic group of muscle atrophies.

Below we present a classification of muscle atrophies, which is based on the international classification of neuromuscular disorders:

1. Muscular atrophies of spinal origin and other dysfunctions of spinal motor neurons: syringomyelia, amyotrophic lateral sclerosis, poliomyelitis, spondylotic myelopathy, spinal muscular atrophies of genetic origin, and others.

2. Muscular atrophies due to peripheral nerve disorders: polyneuropathies (alcoholic, diabetic, nutritional, toxic) , mononeuropathies (traumatic, ischemic, infectious).

3. Muscular atrophies due to primary muscular disorders: progressive muscular dystrophy, polymyositis.
4. Muscular atrophies due to nerve root disorders (may be accompanied by amyotrophy).
5. Other groups of neuromuscular diseases that are not accompanied by amyotrophy, the most important of which is that produced by disorders of neuromuscular transmission at the level of the motor plate and which is characterized by marked muscle weakness, related to physical exercise or any muscular activity. Its main representative is myasthenia gravis.

ALTERATIONS OF REFLECTIVITY

SEMIOGRAPHY AND SEMIODIAGNOSTICS

The alteration of deep (proprioceptive) and superficial (nociceptive) reflexes has value not only as an element of topographical localization, but because, together with other pathological facts, it allows a diagnosis.
clinical diagnosis.

Reflections can present the following situations:

1. Be normal, but the patient has a "neurological problem" not yet evident by pure semiology;
   In these cases, the so-called "complementary study methods" will be necessary, which we will see later.

2. Being diminished (hyporeflexia) or abolished (areflexia).
3. Presenting yourself as exalted (hyperreflexia).
4. Have qualitative alterations: dissociation, opendular inversion.

Normal reflexes

Normal reflexes and the technique of obtaining them were studied in volume 1, Chapter 14. Unfortunately, at the onset of numerous organic diseases of the nervous system, reflexes can remain normal for a certain period. In these cases it is common for the patient to be classified as functional, but time will prove otherwise.

In conclusion, the normality of reflexes during the initiation of certain diseases can lead us to a mistake.

Hyporeflexia and areflexia

Before establishing that a reflex is diminished or abolished, it is advisable to repeat the search maneuver several times, comparing one side with the other. Hyporeflexia or areflexia can be found in three circumstances: when the reflex arc is interrupted at any point along its path, when there is a sudden release of pyramidal control and, less frequently, in a certain type of flexion paraplegia.

We must make particular considerations about some reflections:

1. The masseteric reflex is a difficult reflex to obtain. If it is easy to obtain, it will almost always be an exaggerated
2. The pharyngeal reflex may be absent without this indicating disease.
3. Abdominal cutaneous reflexes may be lacking in obese people and in those whose abdomen is distended or too flaccid.
4. The Babinski sign can be obtained in states such as: hypoglycemia and hepatic or diabetic coma, which are potentially reversible, so its presence does not necessarily imply anatomical damage to the pyramidal tract.

There are six tendon reflexes that are generally constant in the normal subject: patellar, Achilles, bicipital, tricipital, stylorradial and ulnopronator. The first four should almost never be missing. The last two sometimes require careful exploration to find.

Osteotendinous or deep areflexia

The abolition of deep tendon reflexes can occur when the reflex is interrupted by some injury, in any of the pathways that comprise it: afferents (peripheral nerves, posterior root, posterior cords) or efferents (motor neuron of the anterior horn, anterior root of the cord, peripheral nerves). These reflexes may also be abolished if the effector organ (muscle) is affected, as occurs in myopathies.

The causes of osteotendinous areflexia, depending on the location of the reflex interruption, can be:

1. Due to injury to the afferent or efferent branches of the reflex arc: neuritis, nerve injury, polyneuropathies. Sometimes there may be hyporeflexia.
2. Due to injury to the posterior roots and posterior cord: tabes dorsalis, pernicious anemia, Friederich's disease, syringomyelia.
3. Due to injury to the cell body of the motor neuron of the anterior horn of the spinal cord: acute anterior poliomyelitis and Aran-Duchenne syndrome (clinical form of amyotrophic lateral sclerosis or motor neuron disease).
4. Due to impossibility of muscle contraction. In myopathies there is generally hyperreflexia, but there may be areflexia when the muscle is completely atrophied.

Patellar or patellar areflexia

This reflex is absent in tabes (Westphal's sign), in L4 root compression, in some cases of trichinosis, and in Addie's syndrome; and it is exalted in lesions of the pyramidal tract and in anxiety neurosis. The reflex is pendular when the leg describes a series of oscillations after being hit and before remaining motionless. This is seen in cerebellar hypotonia and chorea.

Achilles areflexia

This reflex is abolished in tabes dorsalis, radicular sciatica due to compression of S1, diabetes and myxedema.

Outside of these cases, there may be areflexia in stroke apoplecticus, during the coma phase, where there is a decrease or abolition of reflexes on the hemiplegic side (preceding the frank hyperreflexia that will occur later) and in complete spinal sections, whether functional (spinal cord shock) or anatomical (traumatic section, transverse myelitis).

Precisely in these cases, the examination of the reflexes serves to differentiate whether the spinal section is anatomical and complete, since when this occurs there will be no recovery of the reflexes, while if the section is functional, they may reappear. This element and the recovery of sensitivity are used as prognostic factors in cases of functional spinal cord section.

There may also be a decrease or abolition of reflexes in cachexia due to chronic diseases, such as cancer or Addison's disease.

The temporary disappearance of the patellar reflex has been observed in cyclists immediately after a race, as well as after long walks.

Superficial or cutaneousmucosal areflexia

Cutaneomucosal reflexes are abolished or diminished, also in lesions of the elementary reflex arc, such as in neuritis, radiculitis and tabes;
But since these reflexes also hypothetically have a superior cerebromedullary arch, the lesions that affect the latter also determine its decrease or abolition.

That is why the abolition of the cutaneous-addominal, cremasteric and plantar reflexes is a rule in subjects with pyramidal lesions (hemiplegia and paraplegia) that contrast with deep hyperreflexia. The bilateral abolition of cutaneous-abdominal reflexes is considered quite characteristic of multiple sclerosis.

The isolated abolition of some mucosal reflexes can be verified by:

1. Abolition of the corneal reflex. If the reflex does not occur even though the subject perceives corneal contact (preserved sensitivity), the lesion resides in the motor branch (peripheral facial paralysis), since in central paralysis the reflex persists. If the lesion is located in the sensory branch, the patient does not notice the contact and the reflex, therefore, it does not occur due to the lack of a triggering sensory stimulus. Prolonged use of diazepam decreases or abolishes the motor response (Baird, Pileggi).

   It frequently disappears bilaterally in hysteria.

   More important is the verification of its unilateral disappearance, which is an expression of a lesion of the trigeminal or facial nerve, which constitute the afferent and efferent branches of the reflex. Unilateral loss or disappearance of the corneal reflex is often the first clinical sign of trigeminal paresis, long before any objective sensitivity disorder can be proven. It is observed in tumors of the cerebellopontine angle.

   Its disappearance can also be seen in neuritis of the facial and some branches of the trigeminal nerve.

   The corneal reflex is also abolished in deep comas. This abolition is bilateral and is related to the depth of the coma, which is why it is used as an important element to specify its degree.

2. Abolition of the pharyngeal reflex. It is commonly observed in hysteria and pneumogastric conditions.

Hyperreflexia

Hyperreflexia is the exaggeration of reflexes. We are going to refer especially to osteotendinous or deep hyperreflexia. When it is pathological, it is due to injury to the first neuron or pyramidal neuron, anywhere along its path. The pyramidal pathway exerts a control or inhibition effect on reflex activity, which is a property of the second motor neuron or motoneuron of the anterior horn of the spinal cord. Its injury would produce the release of said control, giving rise to hyperreflexia that manifests itself when exploring the osteotendinous spinal cord reflex activity.

Injury to the pyramidal tract. In organic hemiplegia, hyperreflexia is only on the paralyzed side; In multiple sclerosis, hyperreflexia is generally bilateral;
It also occurs in amyotrophic lateral sclerosis, spastic paraplegia, cerebellar heredoataxia and, more rarely, in pernicious anemia.

Positive plantarcutaneous reflex or Babinski sign

Babinski's sign. It means that the cutaneous-plantar reflex is pathological or positive. It allows us to affirm the organic or functional alteration of the pyramidal pathway; In the latter case (narcosis, uremic and diabetic comas, etc.), it disappears when the cause that motivates it ceases.

Pathophysiological meaning of Babinski sign

Babinski's sign is related to and has the same meaning as spinal automatic reflexes.
Both are pathological reflexes, that is, they never occur under normal conditions. Both are nociceptive, which means that they are produced in response to pain, which often must be provoked in order to obtain them. Barraquer said that pathological defense reflexes are not actually new reflexes, but only the result of the breakdown of the harmonious integration of the flexion and extension components of the normal reflex mechanism. The most important consequence of pyramidal disorder is that the pathological reflex dominates most of the receptive field.

Clonus

Concept and semiography

Clonus or clonus (from the Greek klonos: agitation) consists of a series of involuntary rhythmic contractions, determined in a muscle group by the sudden and passive extension of the tendons; For clonus to occur, it is almost always necessary that tendon stimulation be continuous. Clonus is constituted, in a certain way, by a rhythmic series of tendon reflexes. It differs from the reflex in that the excitation or stimulation is prolonged, in such a way that, having finished the first contraction, a second contraction is immediately produced, and so on, until what is called inexhaustible clonus. It can be inhibited by any peripheral excitation, exerted at a point far from where the clonus occurs. For example, a pinch in the thigh can immediately stop a foot clonus.

Semiogenesis or pathophysiology

Clonus represents, according to its pathophysiology, a hyperexcitability of the reflex arc, due to suppression of the braking or regulatory action normally exerted by the pyramidal pathway. It is mainly observed in patients with enhanced deep reflexes.

Semiotechnics

Clonus of the foot

The doctor instructs the patient to bend his leg on the thigh and rests it on his forearm, takes the foot with his full hand or rests his palm on its plantar surface and performs a passive forced flexion of it, keeping it in that position; Then a series of rhythmic shakes begin. The pathological clonus is frequently inexhaustible and ends with passive extension of the big toe. The non-pathological clonus is soon exhausted (Fig. 51.1).

Clonus of the patella

With the limb extended, take the kneecap between the index fingers and thumb of one hand and make a quick downward movement, as if you wanted to suddenly touch the foot with it, keeping it in that position; If positive, clonus occurs. It is observed in the same cases as clonus of the foot (fig. 51.2).

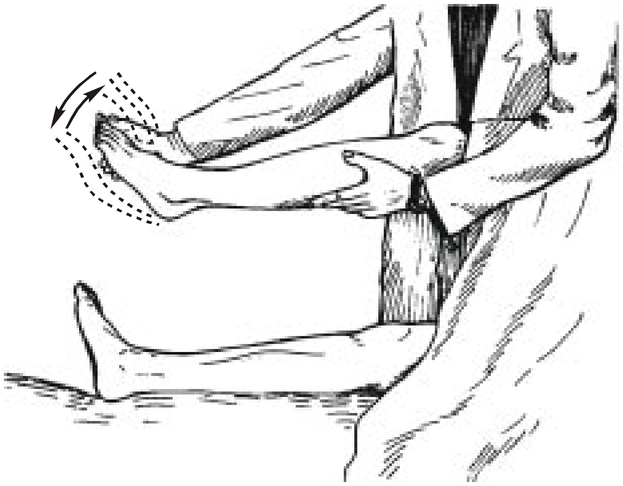


Fig. 51.1 Clonus of the foot.

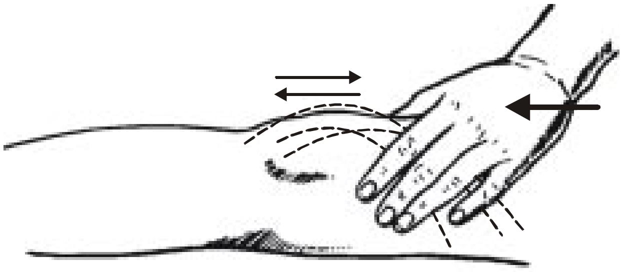


Fig. 51.2 Clonus of the patella.

MUSCLE TONE ALTERATIONS

SEMIOGRAPHY AND SEMIODIAGNOSTICS

Hypertonia

It is the increase in muscle tone. It is generally due to lesions located in the extrapyramidal pyramidal system before its fibers reach the motor neuron of the anterior horn of the spinal cord. Hypertonia should include extrapyramidal rigidity, spasticity of pyramidal origin, decerebrate rigidity, and catatonic rigidity. Hysterical rigidities obey a psychogenic mechanism.

Extrapyramidal rigidity predominates in the muscles of the face, neck, and proximal regions of the limbs; In the exploration of the passive motility of the latter, when flexing the forearm on the arm, for example, we observed that it is carried out gradually, in jumps, as if at the level of the elbow there were, instead of a joint, a gear made up of two toothed wheels. This alteration is known as the cogwheel sign or Black sign.

Pyramidal spasticity predominates in the area or part of the body affected by the injury, that is, more or less paralyzed. It affects one or more extremities of the body, without affecting the muscles of the face or neck. It is considered related to the predominance of the activity of the gamma motor neurons of the anterior horn of the spinal cord, since when the pyramidal pathway is interrupted, the activity of the alpha motor neurons would cease or considerably decrease.

The spasticity or hypertonia of myotonia is related to disorders of the muscle itself; and those of tanos tea and hypocalcemia, with dysfunction of the anterior horn of the cord.

Hypotonia

Hypotonia is decreased muscle tone;
It often accompanies amyotrophies, so its classification is very similar to that presented in the section dedicated to trophism:

1. Hypotonia of spinal origin: anterior horn lesions, poliomyelitis, Werdnig-Hoffmann type muscle atrophy.
2. Hypotonia due to peripheral nerve disorders: polyneuropathies.
3. Hypotonia due to lesions of the roots of the spinal cord: disc compression, tabes dorsalis, spinal anesthesia and spinal tumors.
4. Hypotonia due to primary muscle disorders: muscular dystrophies.

Dystonia

It is an alteration of tone, sometimes with stages of hypotonia or hypertonia, or both at the same time.

AL TERATIONS OF MOTILITY

SEMIOGRAPHY AND SEMIODIAGNOSTICS

Paralysis. It is the impossibility of actively carrying out the movement that corresponds to a muscle or group of muscles and is due to a functional or organic alteration at any point of the pyramidal pathway, from the cor- cerebral complexion to the muscle.

Paresis. From the Greek parens: laziness. It is a lower degree of disability than paralysis; In it it is possible to partially carry out the movement of a muscle or group of muscles; In this case, the movement is slower, or less harmonious. Its cause is the same, but the alteration does not totally damage the neuromotor complex.

If the paralysis reaches a limb (whether upper or lower) we speak of monoplegia (brachial, in the upper limb; crural, in the lower limb). If the paralysis If it takes half the body we talk about hemiplegia (right or left), which is named according to the affected side. If two homologous limbs are affected, this condition is called paraplegia (brachial or crural); If all four limbs are affected it is called quadriplegia.

Functional impotence. It is the same as paralysis or paresis, but the cause is an osteoarticular process that prevents mechanical movement. Example: whoever has ankylosis (welding of the joint surfaces) of the knee will not be able to flex the leg on the thigh, but this lack of movement of the limb is not paralysis, but functional impotence.

Paralysis due to injury to the central motor neuron and injury to the peripheral motor neuron

Paralyses can be due to injury to the upper neuron or first neuron (which runs from the cerebral cortex to the anterior horns of the spinal cord or their equivalents in the brainstem) or to injury to the lower neuron or second neuron (which runs from the anterior horns of the spinal cord, or their equivalents in the brainstem, to its final destination).

We will study them together, pointing out their differences (Table 51.1). We must take into account what we have explained before regarding the trophism, tone and pathway of motility from the peripheral motor neuron (final common pathway of motility) (Chapter 14, volume 1) and remember that injury to it or its effector pathways (roots and nerves) affects all forms of pyramidal and extrapyramidal motility.

PARTICULAR STUDY OF HEMIPLEGIC: HEMIPLEGIC SYNDROME

Ways to start

As we noted, hemiplegia can occur in minutes, a few hours, or over the course of several days. In all cases, this form of onset of hemiplegia and all the accompanying symptoms must be perfectly specified. The sudden onset form constitutes the so-called apoplecticus stroke, which is frequently accompanied by alterations in the level of wakefulness and often leads to coma.

Recognition of hemiplegia during coma

1. Total loss of consciousness (coma): the head and eyes facing the brain injury, that is, diverted in the opposite direction to the paralyzed side. It may be missing in some cases.
2. Facial asymmetry: the face appears wider on the paralyzed side, in which the cheek is seen to rise with each expiratory movement and depress with each

Table 51.1

Original entral paralysis	Paralysis scori gnprif ério
1. Generally it covers many muscles. Never to one Sun or	1. You can take lean muscles
2. When the injury is established there may be sagging, but later there will be hypertonia or contracture of the paralyzed muscles	2. There will be permanent hypotony, atony and weakness of the paralyzed muscles of the left side.
3. There is no true muscle atrophy, there is only a decrease in the volume of the muscle group affected by the lack of newness, that is, by disuse	3. There is early and marked atrophy of the paralyzed muscles (or muscles)
4. The osteotendinous reflexes of the affected muscles are preserved or exaggerated, even though at the time of onset of the paralysis they could be temporarily abolished	4. The osteotendinous reflexes of the affected muscles are diminished or abolished
5. Abdominal cutaneous reflexes are very different or abolished	5. Abdominal cutaneous reflexes are normally preserved
6. There is a Babi nski sign	6. There is no Babi nski sign
7. If the reaction is explored it will prove that there is no reaction electrical degeneration of the same	7. In the exploration of the electrical reactions, a ratio of degeneration or denervation will be obtained

inspiratory movement constituting the sign of the " pipe smoker".

3. Miosis of the pupil on the paralyzed side.
4. Absence of the corneal reflex on the paralyzed side.
5. When compressing the posterior edge of the ascending ramus of the lower jaw, which produces pain, it is observed that only the muscles on the healthy side of the face contract. It's the Pierre-Marie-Foix maneuver.
6. If both lower or upper extremities are lifted simultaneously and dropped onto the bed, the one that falls faster and heavier corresponds to the paralyzed side.
7. Deep tendon reflexes: areflexia or hyporeflexia will be observed on the paralyzed side.
8. Abdominal cutaneous reflexes: they will be absent on the paralyzed side.
9. Babinski sign: will appear on the paralyzed side; less frequently it is bilateral.

Characteristics of hemiplegia outside of coma We can find three varieties:

- Flaccid hemiplegia.
- Spastic hemiplegia.
- Larvate hemiplegia.

Flaccid hemiplegia

We have already described something of this hemiplegia when speaking of the attitude in bed. If the patient manages to come out of the coma, in the following days he will present:

1. A facial paralysis of central origin, that is, inferior type facial paralysis in which the muscles of the forehead, the superciliary and the orbicularis oculi are affected but less than those in the lower territory of the facial nerve, which are totally paralyzed.
2. The patient can occlude the eye on the paralyzed side (which is opposite to the cortical location of the lesion), simultaneously with the eye on the healthy side but not in isolation. This is because each cortical center of the facial nerve supplies innervation to the pontine nuclei of the superior facial, that is, each cortical center of the facial sends a thicker bundle of crossed fibers to the nuclei of the superior and inferior facial on the opposite side and a thinner bundle of direct fibers to the nucleus of the superior facial on the same side. So the upper nucleus of the facial receives crossed and direct fibers (that is, from both hemispheres) while the lower nucleus only receives crossed fibers (from the opposite side). Consequently, a lesion at the level of the cerebral cortex or the internal capsule, on the left side, will affect the crossed fibers (thicker bundle) that go to the nuclei of the facial nerve (superior and inferior) on the right side. But the right upper nucleus has direct fibers (thinner bundle) that it has received from the cortical center of the facial on that side; Therefore, in order to occlude the right eye, it uses its direct corticonuclear fibers and the cortical center of the facial on the right side is also put into action, but as this at the same time, through its crossed fibers that are intact, innervates the orbicularis oculi on the left side, said muscle will contract simultaneously with the one on the right side.
3. If we order the patient to open his mouth and stick out his tongue, we will observe that the tip is deviated towards the paralyzed side due to the action of the muscle.

Genioglossus on the healthy side. The normal action of the genioglossus muscle is to divert the tip of the tongue to the opposite side.

4. There is flaccid paralysis of the upper and lower extremity on the same side as the facial paralysis.
5. There is abolition or decrease in deep tendon reflexes on the paralyzed side.
6. There is a decrease or abolition of cutaneous-abdominal reflexes.
7. The Babinski sign is checked on the paralyzed side.

Spastic hemiplegia

We have already described its manifestation in the standing attitude (see *Attitude") and in walking (see "March").

1, When observing the muscles on the paralyzed side we see their more marked reliefs, and when exploring their motility we verify the existence of hypertonia or contracture.

2. There is exaggeration (hyperreflexia) of the Osteotendinous or deep reflexes on the paralyzed
3. The abolition or decrease of cutaneous-abdominal reflexes is maintained.
4. Clonus appears in the foot and patella on the paralyzed side.

5. Babinski's sign remains present.

The set of symptoms: hypertonia, hyperreflexia, clonus and presence of the Babinski sign, constitutes pyramidalism syndrome, so called because it is considered to be characteristic of a lesion in the pyramidal motor pathway.

Larvate hemiplegia

Also called pyramidal deficiency syndrome. Its discovery or recognition is of great importance because it sometimes precedes, for some time, a pyramidal syndrome itself, but of a destructive type. Certain maneuvers are then required to demonstrate the motor deficit, which are generally not necessary in well-established hemiplegia.

Barré and Mingazzini maneuvers. They were already described in volume 1 (see text and figures in Chapter 14).

Finger separation maneuver, by Barré. The patient places both hands, one in front of the other, so that they "look at each other" without touching each other's palmar surfaces, with the fingers as far apart as possible; The hand on the affected side is the one that can least separate them or the one that tires the fastest (fig. 51.3).

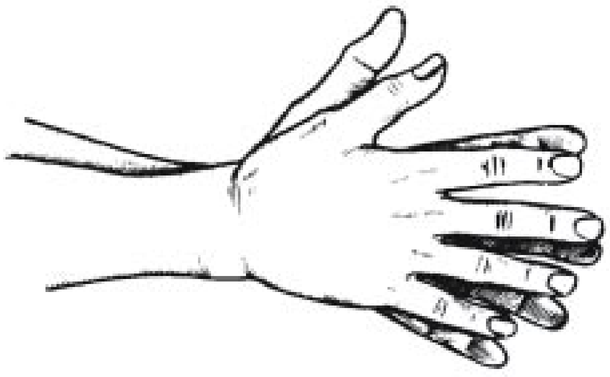


Fig. 51.3 Maneuver of separation of the fingers of the hands, by Barré.

Classification of hemiplegia

If the face and both limbs are affected, it is said to be total.

If the face and the two affected limbs are on the same side, it is symmetrical or direct.

- If the face and both limbs are affected in a more or less equal intensity, it is said to be proportional.

If the affected face corresponds to one side of the body and the two limbs to the other, it is said to be alternate.

Topographic diagnosis of hemiplegia (diagnosis of the height of the lesion)

Cortical hemiplegia

The lesion generally does not cover the entire cortex, that is, the entire height of the motor area, so the paralysis is generally not proportional, but rather one part of the body is more intensely affected than another, potentially producing monoplegia instead of hemiplegia.
The sensory pathway (isolated at this level) is not injured, which means that there are few sensitivity disorders.
Focal epileptic seizures (Jacksonian type) frequently occur that begin partially, always in the same place, and then become generalized; For example, a crisis begins with contractions of the right frontal, the right orbicularis oculi, then the right arm and the right leg. Alterations in intellectual functions are also seen.

If the brain injury lies in the dominant hemisphere (generally the left for all those who are not left-handed), intellectual disorders will be added to the understanding and expression of the heard or written word, which is known as aphasia; and disorders or difficulty in carrying out more or less automatically, but in an orderly and successive manner, certain habitual acts whose objective is to fulfill or carry out a purpose or purpose.

specific objective, such as lighting a cigarette, combing one's hair, brushing one's teeth, which is known as apraxia.

Subcortical hemiplegia

Let us remember that the cortical fibers gather very close to their cortical emergence in a fan-shaped bundle (the corona radiata). From this it is deduced that subcortical lesions have a tendency to take more fibers than cortical ones and although the manifestations predominate in one limb, the rest of that side always participates with a certain degree of deficit. Therefore, monoplegia is rare in the members and convulsive seizures like Jacksonian epilepsy are absent.

Capsular hemiplegia

It is what we have described when referring to hemiplegia during coma, which can take on a flaccid or spastic form depending on the time of evolution. It is possibly the most frequent location in the clinic.

Thalamic hemiplegia

In these cases the hemiplegia is blurry, not very evident, since the pyramidal bundle is only affected by the neighborhood. The contracture is often missing. Come back quickly. Babinski's sign is usually absent. Very important for its diagnosis are the signs dependent on sensitivity, which are light with regard to superficial sensitivity (touch, pain and temperature) but are very intense with regard to deep sensitivity (see sensitivity below) and as a consequence the patient presents hemiataxia (see, below, motility, taxiy or coordination).

Very important for the diagnosis of this rare variety of hemiplegia is the presence, on the paralyzed side, of paroxysmal pain that is very resistant to treatment (central pain).

Truncular and bulbar pyramidal syndromes

This is a group of syndromes described by neurologists and clinicians of the 19th and early 20th centuries and whose identification demonstrates the extent to which deep knowledge of anatomy allowed them to reach accurate diagnoses. Almost all are named after the doctors who described them.

The characteristics of these syndromes are:

1. There are clinical manifestations on the same side of the lesion that are due to lesions at the level of the nuclei or the cranial nerves or both, which "point like a finger to the anatomical site of the lesion."

2. At the same time, clinical manifestations occur on the opposite side of the injury, due to damage to the airways. long that drive motility or sensitivity, or both at the same time.

3. Its etiology is due in most cases to vascular lesions (linked to the vertebrobasilar and posterior cerebral system), tumors, toxic or infectious; and less frequently degenerative.

It is worth remembering that the pyramidal bundle that starts from the cortex, crosses the oval center, reaches the internal capsule and continues its journey without crossing to the lower part of the medulla oblongata (decussation of the pyramids); but the geniculate bundle that also forms the motor pathway, once it passes the internal capsule and enters the peduncle, begins to give its fibers to the motor cranial nerves that it encounters on its descent, always crossing the midline to give it to the nerve on the opposite side.

In this way, once we enter the brain stem (peduncle, pons and medulla oblongata) we find the pyramidal bundle without crossing and cranial nerve nuclei that already have their motor fibers coming from the geniculate bundle on the opposite side.

It will be easily understood that any injury that affects the right or left part of the peduncle, pons or medulla oblongata will produce a paralysis on the same side of the cranial nerve located there (since the nucleus has already received its fiber before) and also a hemiplegia, but on the opposite side, because the injured pyramidal tract has not yet been crossed.

These clinical syndromes can be included in the so-called alternating hemiplegias.

In summary we point out that:

a) all alternating hemiplegia is constituted by paralysis of one or more cranial nerves on the same side as the lesion and hemiplegia on the side opposite to the lesion, and other structures of the stem may be affected, which will sometimes produce complex clinical syndromes (see brain stem syndromes: peduncular, pontine and bulbar);

b) the clinical varieties depend on the site where the lesion occurs, covering in that place the pairs included therein (hence its topographic value to locate its height);

c) that for alternating hemiplegia of any variety to occur, it is an essential requirement that the lesion be located in the brain stem, that is, below the internal capsule;

d) that any lesion located in the internal capsule above it will produce direct hemiplegia.

Topographic location of truncal and bulbar pyramidal syndromes

1. Alternating peduncular hemiplegia (Weber syndrome) (Fig. 51.4) . It affects the pyramidal bundle in its peduncular path and the fibers that have just left the nucleus of the III pair and run through the interior of the cerebral peduncle. It is constituted by complete hemiplegia on the side opposite to that on which the lesion occurs, with the lower facial, upper and lower limbs on that side being taken, and complete or partial paralysis (remember the multiple nuclei of origin of the III nerve) of the oculomotor motor on the same side on which the lesion is located, that is, on the opposite side to that of the paralysis of the limbs. This paralysis of the III nerve may give, depending on the case, ptosis of the upper eyelid of the affected eye and deviation of the eye on the affected side outwards (due to paradysis of the internal rectus muscle of the eye that remains without counteracting the action of the external rectus muscle, innervated by the VI nerve).

2. Alternating pontine hemiplegia (Millard-Gübler syndrome; lesion at the level of the bridge foot). This lesion affects the pyramidal pathway before crossing and the nuclei of the VII nerve and sometimes the nucleus of the VI nerve that is surrounded by the fibers of the VII nerve at its origin. The result will be: hemiplegia on the side opposite to that of the injury (taking upper or lower limb but without taking the face on that side) and a peripheral facial paralysis (which covers the entire face, both the upper and lower), with or without paralysis of the VI nerve (abducens) on the same side as the facial (fig. 51.5).

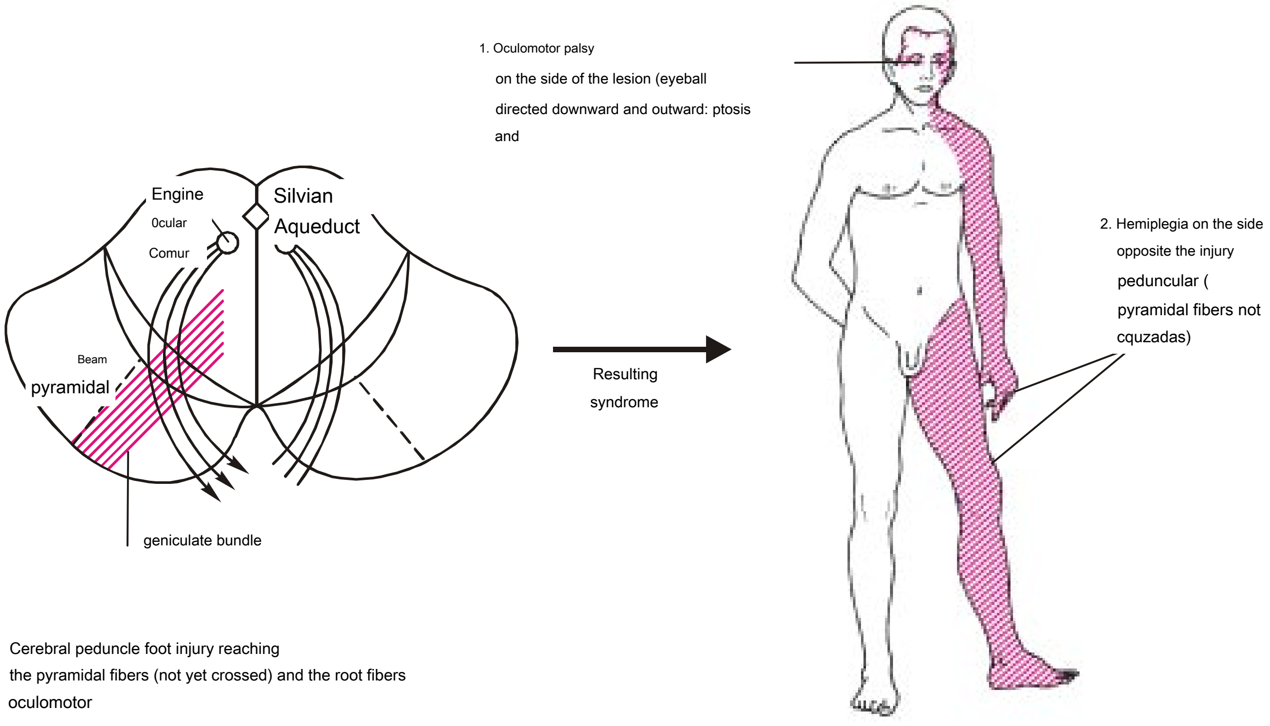
3. Alternating bulbar hemiplegia. The lesions that produce it affect the pyramidal pathway and the sensory pathway before crossing and the nucleus or roots, or both, of the V (sensory root), LX, X, XI, and XII pairs already crossed. Depending on the height of the lesion in the medulla oblongata, injury to one or more cranial nerves will occur, causing the different syndromes of alternating bulbar hemiplegia. Furthermore, in certain cases due to injury to intrabulbar sympathetic elements, Claude-Bernard-Horner syndrome appears (on the same side as the injury).

4. Fibers that carry impulses to the cerebellum and give rise to elements of the cerebellar series (vertigo, lateropulsion and ataxia on the same side) may also be affected (on the same side as the lesion).

ATAXIA

From the structures that intervene in the coordination mechanism (described in Chapter 14, volume 1) it is deduced that an ataxia may be produced by:

Fig. 51.4 Alternating peduncular hemiplegia (Weber syndrome).


1. Oculomotor palsy
on the side of the lesion (eyeball
directed downward and outward: ptosis
and
Engine
Ocular
Comur
Silvian
Aqueduct
Beam
pyramidal
geniculate bundle
Resulting
syndrome
2. Hemiplegia on the side
opposite the injury
peduncular (
pyramidal fibers not
cquzadas)
Cerebral peduncle foot injury reaching
the pyramidal fibers (not yet crossed) and the root fibers
oculomotor

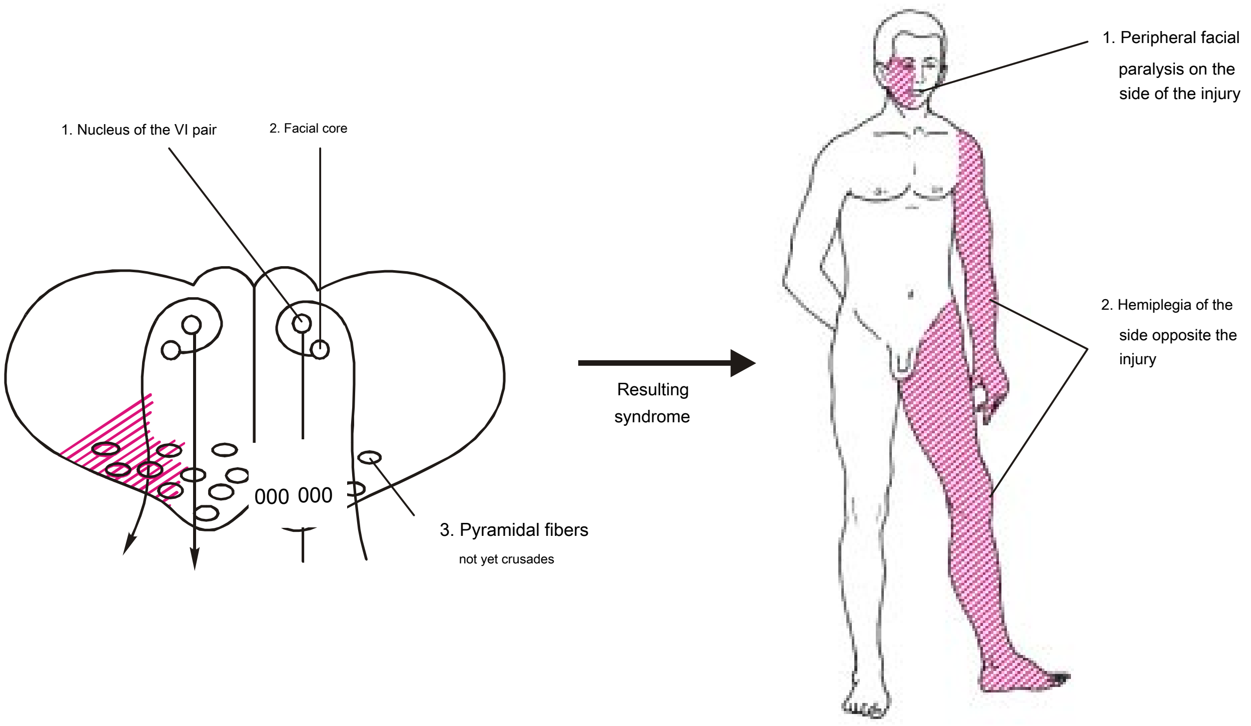


Fig. 51.5 Pontine alternating hemiplegia (Millard-Gübler syndrome).

1. Lesions or dysfunctions of the cerebellum or its main connections.
2. Peripheral nerve injuries.
3. Injuries to the posterior cord of the cord.
4. Injuries or dysfunctions of the labyrinth, nerve and vestibular pathways.
5. Less frequently, lesions of the cerebral cortex.

ALTERATIONS OF STATIC COORDINATION

Semiological importance of Romberg's sign

As explained in detail in Chapter 52 (see VIII cranial nerve) , this sign reveals a deficit in the conduction of proprioceptive stimuli through the pathways of deep sensitivity or the vestibular pathways, which is corrected thanks to the sense of sight;

By canceling the latter by making the patient close his eyes, the ataxic disorder is clearly evident. For this reason, Romberg's sign mainly occurs in two types of lesions: those that affect the deep sensory pathways of the spinal cord (for example, tabes dorsalis) and those that affect the vestibular pathways.

ALTERATIONS OF DYNAMIC COORDINATION

Semiography and semidiagnosis

1. Peripheral ataxia. This is the name given to ataxia caused by lesions of the peripheral nervous system, such as polyneuritis.
2. Spinal ataxia: Lesions of the posterior cords of the spinal cord that affect proprioceptive sensitivity, examples: tabetic ataxia, combined sclerosis ataxia. In both peripheral and spinal ataxia, the patellar reflex is absent.
3. Cerebellar ataxia. It is another of the best defined ataxias and is found in the cerebellar syndrome. There is no Romberg sign and there are other differential features with tabetic or spinal ataxia, particularly the symptoms of cerebellar syndrome (see Chapter 54).
4. Labyrinthine ataxia. It is typical of the labyrinth processes. Auditory phenomena are usually associated with it: vertigo, tinnitus, and Romberg's sign appears after a short interval in which the patient remains upright. There is nystagmus.
5. Thalamic ataxia. In lesions of the optic thalamus, hemiataxia occurs on the affected side. Certain lesions of the parietal, temporal and frontal cortices can cause

ataxia, such as the so-called Bruns frontal ataxia, which accompanies tumors of the frontal lobe of the brain.

6. Mixed ataxias. They depend on both peripheral and central lesions, such as the ataxia observed in plaque sclerosis and acute ataxia due to disseminated (multifocal) myelitis.

AT TERATIONS OF PRAXIA

Praxia and its exploration were already briefly addressed in Chapters 13 and 14 of volume 1. In this chapter we will address it in detail.

According to Monrad-Krohn, human beings only have three mechanisms to express their thoughts. So Kant's " faculty of expression" would be given through:

1. The spoken word.
2. The written word.
3. Mimicry, gestures and actions.

These psychomotor functions are translated respectively into three forms of psychomotor reaction used by a person to respond to different tests. They are: the verbal response, the written response and the ideokinetic response in the form of actions:
gesticulations, gestures and mimic movements.

When these expression mechanisms are altered, disorders arise that in the case of the spoken word is called aphasia, in the case of the written word it is called agraphia and in the case of mimicry, gestures and actions it is designated as apraxia.

The psychomotor functions outlined need and are in constant interrelation with psychosensory functions, that is, with auditory perception, visual perception and tactile perception. So an alteration in one or more psychosensory functions has an impact to a greater or lesser degree on psychomotor functions.

DEFINITIONS OF PRAXIA

Praxia or praxis (from the Greek praxis or prassis, to act or prassein, to do or pratto, work) is called the faculty or capacity to execute or fulfill, more or less automatically, organized, complex and intentional voluntary movements, that is, in accordance with a determined plan, learned by previous experience or, in other words, coming from learning.

The motor activity of the human being derives, for the most part, from learning. In this way, they create motor burns that are stored from the repetition of certain actions integrated by movement systems. These habitual movements directed toward a specific goal are complex motor acts in which the successive simultaneous and regular contraction of several muscle groups participate, which, as already stated, arise in the process of learning and imitation in the relationship of the individual with the environment.

Among these psychomnotor acts, as they have been called, or gestures, are: combing one's hair, lighting a cigarette, brushing one's teeth, saluting, taking a coin out of one's pocket, making the sign of the cross, saluting the flag, and others.

Liepman has differentiated two types of acts: intransitive ones, which are carried out without the intervention of any object (saying hello, making the sign of the cross) and transitive ones, which are carried out with the intervention of objects (brushing one's teeth, combing one's hair, lighting a cigarette and smoking).

These acts of a certain complexity are made up of simpler acts that require an order of execution, a plan. If they are carried out correctly, we speak of normal praxia or eupraxia. Alterations in this capacity range from a mild degree or dyspraxia to a frank disturbance or apraxia.

For educational purposes and with a view to a better understanding of the concept of apraxia, we offer below some definitions proposed over time by various authors.

The inability to perform skilled movements (rather than language) where extensive motor, sensory, ataxic mental disorder is not detected (Liepmann, 1900; Denny-Brown, 1958).

The impossibility of carrying out movements adapted to a purpose or actions, being neither paralytic, nor insane, nor agnostic' (Dasseny Fustioni, 1945).

Disorder of gestural activity that appears in a subject whose apparatus for executing the action is intact (absence of paralytic, ataxic, choreoathetotic disorders) and who has full knowledge of the act to be performed" (Ajurriaguerra and Hecaen cited by Barraquer-Bordes, 1970).

"Apraxia is characterized by the loss of habits acquired in the process of individual experience for the performance of intentional complex motor actions (domestic, work, symbolic gesticulations) without obvious signs that attest to the presence of a central paresis or disorders of the coordination of movements" (Bogorodinski, Skoromets and Shvarev, 1979).

"Apraxia is the loss of the ability to execute organized voluntary movements, learned through previous experience, without this disorder being attributable to weakness, incoordination, abnormal movements of an extrapyramidal type or any state or dysfunction of the nervous system that makes it impossible to perceive and understand orders. Therefore, what is lost is the motor plan or schemes that make up the movements" (Terrazas Hontañón, 1997).

This last concept seems very complete to us and is derived in practical terms from Terrazas Hontañón himself, that the fundamental characteristic of apraxia is the conservation of the ability to perform isolated movements that make up a motor action along with the impossibility of planning and executing them in an adequate sequence that translates into a precise act.

It is important, when reviewing these concepts, to keep in mind that to define apraxia itself, other alterations must be excluded, for example: agnosia or paralysis or paresis, which would prevent the execution of an act or gesture without an apraxic disorder per se, as we will clarify in more detail in semiogenesis.

CLASSIFICATION

Although a whole variety of apraxias grouped into classic forms and special forms have been described in medical literature, currently ideomotor and ideatory apraxias remain as apraxias per se.

Ideomotor apraxia is the most frequent and the prototype of apraxias, so when the term apraxia without a surname is used it is understood that it is the one referred to.

The patient who suffers from this disorder presents great difficulty or even the impossibility of executing a motor action to a verbal command although he is able to recognize its symbolic value, identify whether it is carried out correctly or not by the examiner and can sometimes describe it.

It is as if there were a dissociation between the idea or plan of movements, generally preserved, and the realization, frankly disturbed.

It is very important to be certain that the patient has understood the order, since if not, the patient, instead of an apraxic, would be an aphasic.

Ideatoria apraria is the other form of classical apraxia that, despite there being no unified criterion for it, is recognized as an independent type. It has been defined as a disorder of the plan of movement to the performance of these complex acts, in which sequences of more basic movements participate. Therefore, serial act tests are the ones of choice for diagnosis.

Other authors insist that the most relevant peculiarity of ideational apraxia lies in the inability of the patient who suffers from it to use real objects. However, Heilman considers that the most selective test is pantomime in response to a verbal command.

So to combine these controversial approaches and avoid possible misunderstandings, it may be convenient, in the opinion of Terrazas Hontaffňón, to qualify the term apraxia by the type of evidence that is altered: ideatory apraxia of serial acts, ideatory apraxia of real objects and pantomime apraxia of verbal commands.

The other classic forms mentioned in the literature, in addition to ideomotor and ideatory, are callosal apraxia and limb-kinetic or innervatory apraxia. Callosal apraxia is actually a subtype of ideomotor apraxia and limb-kinetics is no longer included in this concept, since a large part of its semiological elements are related to corticospinal dysfunction.

With respect to the so-called special forms of apraxia (constructive, dressing, gait, orofacial, verbal and oculomotor), some researchers suggest that they should not be considered as separate entities. For example, orofacial apraxia can correspond to a topographic location of ideomotor apraxia. There are even authors who question whether some of these special forms of acts are true practices.

SEMIOGENESIS

Voluntary movements (praxis), especially manipulative ones, directed toward a specific goal, constitute a complex functional system. This, according to Luria, brings together a certain number of conditions or factors that depend on the concerted work of a whole group of cortical zones and subcortical structures, each of which makes its own contribution to the realization of the movement.

In the vast majority of cases of apraxia, this is due to lesions of the left hemisphere, which is generally the dominant one. This hemisphere is where both the plan and the movement patterns remain stored. Although it is possible that lesions of the right hemisphere can cause apraxia in left-handed patients, in these patients it occurs with less frequency and intensity, given that they are often ambidextrous and have a greater dispersion of the centers related to praxic function.

More than one neuroanatomical and neurophysiological model has been proposed to explain praxia and, therefore, apraxia. We will briefly see that of Heilman (1979), cited by Terrazas Hontañón, who proposes the existence of an intermediate center for the praxic function, whose fundamental substrate is the supramarginal gyrus, with the possible participation of other adjacent areas of the parietal and superior temporal cortices, and the angular gyrus. This center constitutes a storehouse of visuokinetic motor schemes, which would serve to program sequences of movements, while the premotor area programs isolated movements of more than one muscle.

Following this model, the stages of carrying out an organized voluntary movement in response to a verbal command can be the following:

1. The order must be perceived auditorily and, pardon the redundancy, in the primary auditory cortex (area 41 or Heschl's gyrus) and decoded in Wernicke's area on the left side (auditory association cortex).

2. This information is sent to the supramarginal gyrus and perhaps to the most anterior part of the dominant hemisphere (usually the left), where the words that form the verbal command are associated with a visuokinesthetic memory of the motor scheme. It is in this center where the auditory, visual and somatesthetic stimuli that originate a motor activity in response would be integrated.

3. Information from the parietal center of praxia travels through subcortical white matter pathways from the parietal lobe to the premotor area (area 6) and activates the learned motor schemes.
4. The left premotor area is dominant with respect to the right. The information from the former is sent to the latter through the genu of the corpus callosum.

5. Finally, short association fibers that come from both premotor regions connect with the primary premotor area (area 4) and activate the motoneurons that go through the bilateral pyramidal pathways, which ultimately results in the contraction of the corresponding muscles.

Thus, lesions of the supramarginal gyrus of the dominant hemisphere (usually the left), of the most anterior parietal subcortical region of the dominant hemisphere, of the corpus callosum and of the adjacent white matter can produce apraxia associated or not, depending on the case, with other disorders.

Due to its didactic value, and as a functional and practical counterpart to the neuroanatomophysiological account reviewed, we are going to follow the steps that Dassen and Fustinoni use when exemplifying the study of praxia, with the execution of a transitive act, in which a series of stages must be followed and fulfilled:

1. The first thing is to recognize the object or instrument that you are going to use in the act:
    a) In its elemental qualities (shape, color, consistency, etc.), that is, identifying it.
    b) In its meaning (that is, what it is for).
2. In the case of a patient who is being explored, he or she must first perceive the order we give him and understand it, even before recognizing the object. Here the influence of psycho-sensitive functions (auditory, visual and tactile perceptions) on psychomotor functions becomes manifest in this case: actions and demonstrations.

3. The next thing is: decide its use.
4. Then each of the elementary movements or partial acts necessary for the fulfillment of the global act must be evoked or represented. This series of elemental movements that make up an act constitutes the kinetic formula.
5. Finally, execute the kinetic formula. The latter, of course, having an undamaged motor effector apparatus.

Let's take as an example the act of brushing one's teeth: the person identifies the brush object by its qualities. Then he recognizes its meaning, that is, as an instrument used to clean teeth. Once its use has been decided, it evokes or represents each of the movements that it is going to execute with the brush. That is, take the brush, take it to the mouth, place it on the teeth and direct it towards the different places of the teeth, ranging from the gum to the edge of the teeth, both on the buccal and lingual sides, as well as on the occlusal surface. He will then carry out these movements.

Given the functional complexity of this psychomotor mechanism, in which several stages are involved, the impairment of any of them can disturb the ideomotor response, that is, praxia.

However, it is necessary to distinguish true apraxia from false apraxias. In this way, if the disturbance occurs in the first stage, that is, in the identification of the object, because there is agnosia, the act cannot be carried out.

In agnosia, the patient, although his or her senses (sight, hearing, touch, etc.) are normal, is not able to recognize familiar objects, people or places.
We would then speak of apraxia secondary to agnosia or agnostic apraxia, which is actually a false apraxia.

Likewise, if there is paralysis, paresis or involuntary movements, although the act cannot be performed, we cannot speak of apraxia either.

SEMIOTECHNICS

The exploration of praxia was briefly expressed in volume 1, Chapter 14 and will be further explored here.

From the outset, it is necessary to exclude the existence of agnosias, paresis, paralysis or involuntary movements, which would prevent the act in question without there having to be true apraxia.

To explore praxia and therefore determine if there is any alteration of it, whether to a lesser degree or dyspraxia or to a greater degree or apraxia, the patient is ordered to perform various acts, to observe, first, if he performs them and, second, how he performs them.

Classically there are three types of acts:

1. Intransitives: stick out your tongue, make the sign of the cross, give the military salute.
2. Transitives: light a cigarette, drink a glass of water, take out the watch.
3. Imitative: the patient is asked to imitate the acts that the observer performs.

It is convenient, to evaluate the possible existence of apraxia, to do the tests in a hierarchical order, with a sequence from greatest to least difficulty. That is, first the performance of a gestural or use pantomime in response to a verbal command without the real object. Secondly, the imitation of performing the act without the use of the real object, represented by the doctor. Thirdly, the manipulation of a real object to carry out a certain act.

As you can see, it corresponds to intransitive, imitative and transitive acts.

We will now describe the tests most frequently used to detect apraxias, especially the ideomotor test, as summarized by Terrazas Hontañón.

1. On the extremities:
   a) Gestures: military salute, saying goodbye, ordering someone to come closer, pass or stop.
   b) Manipulative: flip a coin, put out a cigarette, comb your hair, brush your teeth, use a hammer or kick a ball.
2. Orofacial:
   a) Gestures: making the noise of a kiss, whistling, mocking with your tongue, or clearing your throat.
   b) Manipulative: extinguish a flame or drink through an absorbent.
3. Corporal: placing yourself in the position of a boxer or bowing.

These tests must be performed on both hemi-bodies. The alteration in only one of the hemibodies is called hemiapraxia. As we already clarified, they are applied in an order of decreasing difficulty or complexity: pantomime, imitation and finally manipulation of a real object.

In the patient with ideatory apraxia, tests of sequential or serial acts are used, such as telling him to light a cigarette, in which he must: "take the box of matches, open it, extract the match from it, rub the match against the box and light the cigarette.

SEMIOGRAPHY

Let's take, for example, the following command:

"Open a tube of toothpaste, take your toothbrush, and apply the paste to your teeth."

In case of alteration, some of the following facts could be observed:

- The patient does not go beyond the first stages. Let's say you take the tube of toothpaste and hold it in your hand without doing anything else or at most, open the tube of toothpaste.

- The patient tries to do one of the last stages and tries to apply the paste on the brush without having opened the tube or without having removed toothpaste.

Repeats some stages of the act or prolongs them. This is called perseveration, which can be tonic, for example, remaining with the tube open, or even with the toothpaste out, in a fixed attitude. It can be clonic or kinetic perseveration. So, for example, continue applying the paste over and over again on the brush; or an intentional perseverance, when when ordered to apply toothpaste to the brush, he continues, in the previous stage, squeezing the tube again and again to extract the toothpaste.

Another alteration is that with the tube of toothpaste already open, make a gesture other than applying it to a brush, say, the gesture of writing.

In the case of the so-called ideatory apraxia, the patient is not capable of executing the entire sequence, although he is capable of carrying out each part of the order adequately.

SEMIODIAGNOSIS

The injuries that most frequently cause apraxia

Are:

1. Infarction of the area supplied by the left middle cerebral artery (most common cause of ideomotor apraxia).
2. Lesions that affect areas or pathways related to practical function, of traumatic, infectious (abscesses, encephalitis) , neoplastic or hemorrhagic origin.
3. Lesions of the corpus callosum and adjacent white matter

cent:

a) Tumors.

b) Infarctions of the blood distribution area of the anterior cerebral artery.

4. Diffuse degenerative brain disease: Alzheimer's disease.

ALTERATIONS OF SENSITIVITY

SEMIOGRAPHY AND SEMIODIAGNOSTICS

By putting together the set of data obtained in the course of the sensitivity exploration (see volume 1, Chapter 14), different syndromes can be formed:

- Sensitivity dissociation syndromes.

Spinal sensory syndromes.

- Root sensory syndromes.
- Peripheral sensory syndromes.

Sensitivity dissociation syndromes

1. Syringomyelic dissociation. There is abolition of thermal and painful sensitivity (thermoanesthesia and analgesia) and tactile and deep sensitivities have been preserved. It is observed in syringomyelia, a disease that damages the medullary gray matter in its central part, crossed, as we have seen, by the pathways of thermal and pain sensitivity (fig. 51.6). Syringomyelic dissociation is also observed in leprous neuritis. It can also be seen in bridge injuries, when only the fiber bundles that conduct thermal and pain stimuli are affected.

SOs.

2. Tabetic dissociation. It consists of the loss of sensitivity to touch and deep sensitivity, with preservation of thermal and painful sensitivity. It is due to injuries to the posterior spinal cords which, as is known, are made up of the fibers that conduct deep sensitivity and are crossed by the fibers of tactile sensitivity, as they penetrate the spinal cord and go to the gray spinal column. It is observed in tabes (fig. 51.7), although not with the absolute character that could be deduced from this definition, that is, that certain forms of deep sensitivity are only diminished, instead of abolished. This dissociation is also observed in some polyneuritis (pseudotabes polyneuriica).

3. Peripheral dissociation. It is characterized by superficial anesthesia with preservation of deep anesthesia. It generally corresponds to injuries that affect the cutaneous nerves that carry only tactile, thermal and painful fibers.

Spinal sensory syndromes

It is enough to remember the distribution of the sensory pathways within the spinal cord (see Chapter 14, volume 1) to understand the entire series of spinal sensory syndromes that can arise depending on the extent of the lesion in width or height. Fundamentally, the following spinal sensory syndromes are distinguished:

1. Paraplegic sensory syndrome. It is due to lesions that involve the entire width of the cord, that is, that carry out the entire cord section (wounds, trauma, transverse myelitis, tumors, Pott's disease). Absolute anesthesia will be observed, that is.
loss of all forms of sensitivity, the upper limit of which reaches a variable height, depending on the location of the section. If the lesion lies at the level of the cervical cord, sensory disorders can be observed in the face, at the level of the trigeminal territory, because the lesion encompasses the fibers of the root, descending from the V nerve, which, as is known, reach the IV cervical spinal cord segment. There is also a loss of motility (paraplegia), hypotonia and areflexia.
2. Brown-Sequard syndrome (fig. 51.8). If the section affects only half of the spinal cord (hemisection), Brown-Sequard syndrome occurs, characterized by the fact that, in addition to other motor disorders, there is abolition of deep sensitivity on the side of the lesion (abatiesthesia and apalesthesia) and alteration of superficial sensitivity, on the opposite side, which ranges from simple hypoesthesia to anesthesia.
Superficial sensitivity, on the side of the lesion, and deep sensitivity, on the opposite side, are preserved. This is because spinal hemisection injures the already crossed fibers of the superficial sensitivity and the not yet crossed fibers of the deep sensitivity.

Above the upper limit of the lesion, there is a small strip of hyperesthesia; about this one

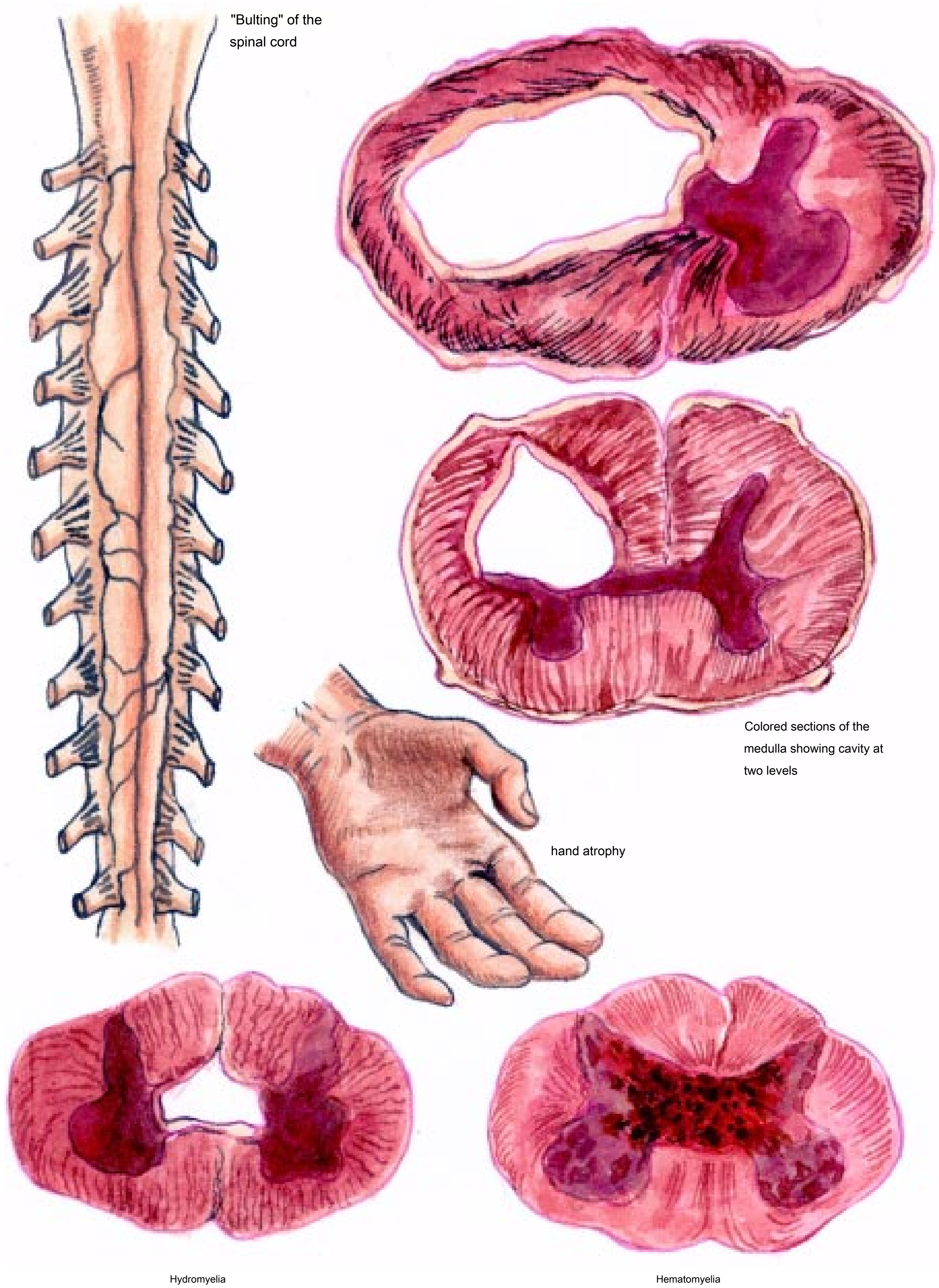

"Bulting" of the spinal cord
Colored sections of the medulla showing cavity at two levels
hand atrophy
Hydromyelia
Hematomyelia

Fig. 51.6 Syringomyelia.

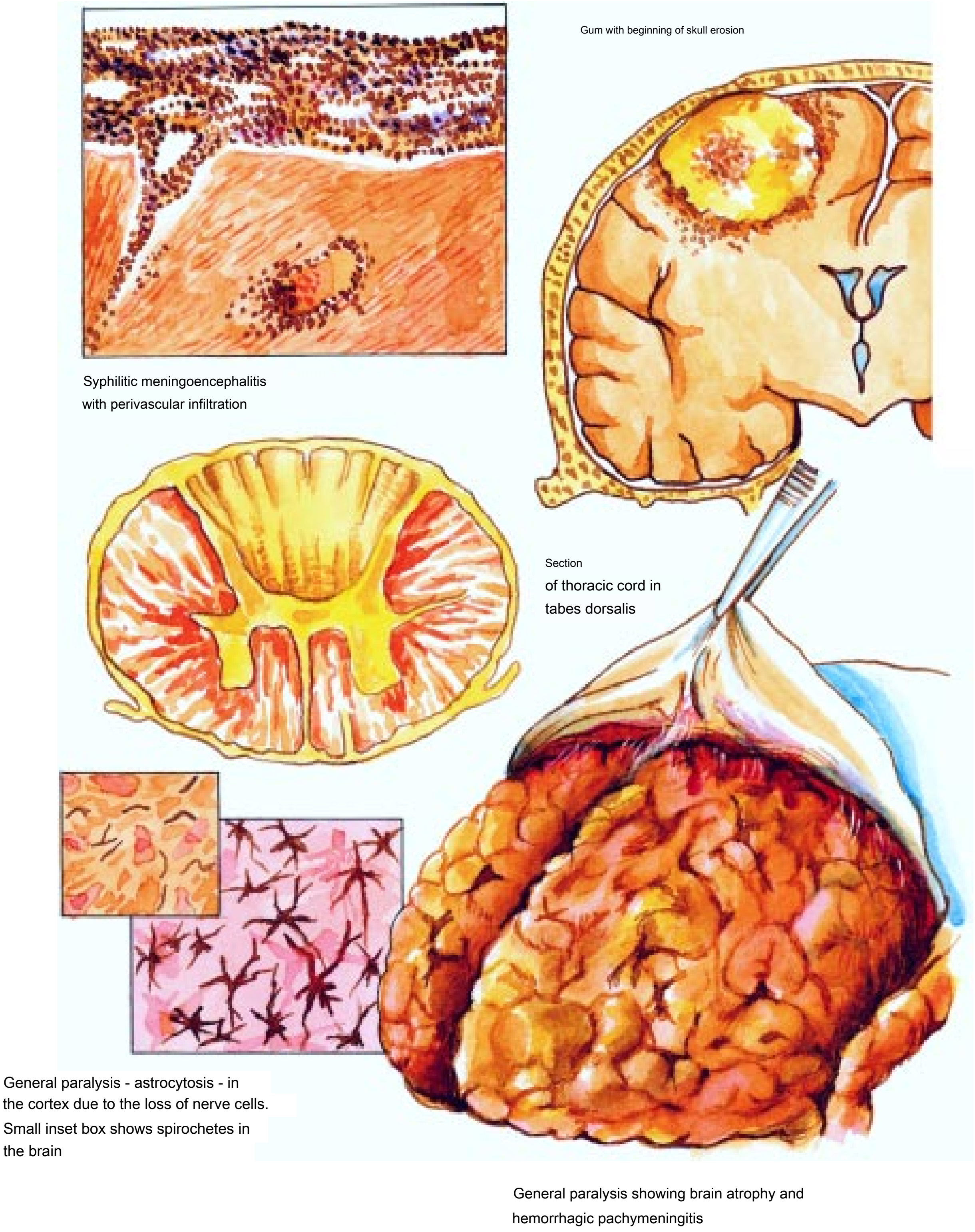
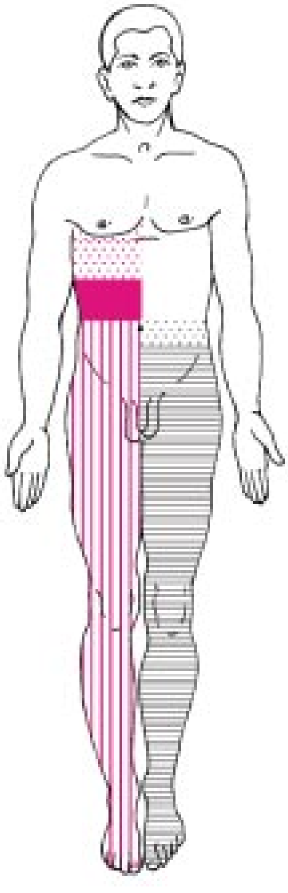

Gum with beginning of skull erosion
Syphilitic meningoencephalitis with perivascular infiltration
Section of thoracic cord in tabes dorsalis
General paralysis - astrocytosis - in the cortex due to the loss of nerve cells. Small inset box shows spirochetes in the brain
General paralysis showing brain atrophy and hemorrhagic pachymeningitis

Fig. 51.7 Tabes.

Radicular type anesthesia area, on which in turn, another band of hyperesthesia is arranged. On the opposite side of the lesion, above the upper limit of superficial anesthesia, there is a band of mild hyperesthesia. These anesthetic and hyperesthetic bands that are arranged at the limit of the lesion are due to the fact that it affects the posterior roots corresponding to the level at which it is located.

3. Posterior spinal cord syndrome (also called Dejerine long root fiber syndrome). The most important alteration is the loss or decrease in vibratory sensitivity (apalesthesia or hypopalesthesia); Abathiesthesia and abaresthesia are equally accentuated. Superficial sensitivity to heat and pain remains intact and

Fig. 51.8 Diagram of Brown-Sequard syndrome produced by the right hemilateral section of the cord at the level of the dorsal region. Side of injury: motor and vasomotor paralysis (red dotted); segmental root anesthesia band for all sensitivities in this area of the trunk (in dark red): loss of deep ipsilateral sensitivity (position, movement and tuning fork) (vertical red stripes). Opposite side of the lesion: loss of painful and thermal surface sensitivity (crossed pathways) (horizontal striping); supralesional hyperesthetic areas (black dotted).



At the beginning of the syndrome, if the examination is rough, so is the tactile sensitivity. However, a careful examination shows that there are objective paresthesias, localization errors and delay of sensations, etc. The syndrome is completed with tabetic type ataxia (posterior cordonal ataxia), that is, both static and dynamic, areflexia or deep hyperreflexia, depending on the lesion that causes the syndrome; In some cases of acute onset, contralateral hyperpathy has been observed, that is, stimuli that are not painful in themselves are perceived by the patient as severe pain.

Causes: neoplasms and trauma limited to the posterior cords, tabes and combined sclerosis of the type of Lichtheim syndrome in pernicious anemia.

4. Sensory syndrome of the anterolateral cords. In this syndrome, intense subjective pain can exist for a long time, with very few objective sensitivity disorders. When the latter are present, they consist of a syringomyelic type dissociation, although sometimes tactile sensitivity may be somewhat affected. The difference with syringomyelia is that in this the disorders are ipsilateral, while in cord lesions they are contralateral.

The pain is due to compression of the posterior roots of the spinal cord. If we remember that the pyramidal, extrapyramidal, spinothalamic and spinocerebellar pathways travel in the anterolateral cord, we will understand the other accompanying symptoms of this particular syndrome (direct) hemiplegia.

Causes: processes that compress the spinal cord from the outside, such as extramedullary tumors, meningeal lesions or bone, tuberculous or tumor lesions.

5. Conus medullaris syndrome The conus medullaris is the lower end of the medulla, which precedes the fillum terminalis; corresponds to the body of the second lumbar vertebra; It gives rise to the last three pairs and the coccygeal nerves that innervate the muscles of the perineum; They receive sensitivity from the skin of the sacrococcygeal region of the anus, perineum and genital organs. In addition, the innervation centers of the bladder and rectum are found in the conus medullaris. Injury to the conus medullaris results in saddle anesthesia, in addition to sphincter and genital disorders.

Radicular sensory syndromes

They include sensory syndromes caused by lesions of the posterior spinal roots (radiculitis) and bone alterations (tuberculous, rheumatic or neoplastic of the spine) that compress the roots, etc. Alterations in subjective sensitivity (radiculalgia) and sensitivity disorders are confirmed.

objective consists of local anesthesia (of all forms of sensitivity, in the territory of distribution of the affected roots; sometimes, instead of total anesthesia, there is tabetic-type dissociation). The distribution of anesthesia is done by bands, which in the limbs are parallel to their axis and in the trunk they are transverse bands as corresponds to the root topography. Let us remember that to observe this band anesthesia, at least three roots in a row must be injured (law of the three roots).

Cauda equina syndrome

The lumbar and sacral roots, before leaving the spinal canal, form a bundle that runs through it, from the second lumbar vertebra (where the spinal cord ends) to the respective exit foramina. This bundle of nerves is called the cauda equina due to its similarity and can be compressed or injured by trauma, meningitis or tumors. An anesthesia similar to that of the conus medullaris is then produced, that is, in the saddle, ano-perineo-scrotal, and there are also anesthesia bands that are arranged along the posterior surface of the thigh and legs; Paralytic, sphincter and genital symptoms are also observed.

The initial pain, as a consequence of the compression of the roots, is important to differentiate this syndrome from the conus medullaris syndrome.

Peripheral sensory syndromes

Under this name, sensory disorders caused by injuries to peripheral nerves (whether exclusively sensory or mixed) and plexuses are studied.

1. Sensory syndromes due to nerve injuries. If the lesion affects a cutaneous nerve, there will be only disorders of superficial sensitivity with preservation of deep sensitivity, that is, peripheral dissociation.
   If the lesions affect nerve trunks, as occurs in polyneuritis or trauma, anesthesia is complete, but with peripheral topography, that is, covering the territory of innervation or distribution of the affected nerves. In some cases, there is only a decrease or delay in the perception of superficial sensations, sometimes more for some than for others.

2. Sensory syndromes due to total plexus injury. In this case there is total anesthesia, which extends to the entire area innervated by the plexus.

NERVE SYSTEM0. COUPLE ALTERATIONS CRANIALS

52

Introduction

The anatomophysiological account and the exploration of the cranial nerves were presented in volume 1, Chapter 15, which it is advisable to review before studying this chapter.

Below we will do a particular analysis of the alterations of the cranial nerves, in the same order used for their exploration.

- Olfactory nerve: I pair.
- Optic nerve: II pair.
- Common oculomotor, pathetic (trochlear) and abducens (abducens) nerves: III, IV and VI pairs.
- Trigeminal nerve: V pair.
- Facial nerve: VII pair.
- Statoacoustic nerve: VIII pair.

Glossopharyngeal, pneumogastric (vagus) and accessory nerves: IX, and XI pairs.

- Hypoglossal nerve: XII pair.

OLFACTORY NERVE: I PAIR

semiography

1. First we will exclude nasal conditions, a common cause of decreased or loss of smell. In these cases it can be bilateral (more common) or unilateral.
2. Intracranial lesions almost never produce alterations in smell unless the olfactory tracts are affected and in this case the lesion produces unilateral anosmia.
3. Bilateral anosmia. Condition of the nasal passages.
4. Unilateral anosmia. Injury to the olfactory tract.
5. HypoSmia. Minor degree of the sense of smell.
6. Parosmia. Confusion of smells; indicates injury to the cerebral cortex.
7. Olfactory hallucinations. Perception of odors without an external odorous stimulus; indicates injury to the cerebral cortex.

Semidiagnosis

1. Anosmia. Tumors of the frontal lobe (lower face) such as meningiomas; fractures of the base of the skull.
2. ParoSmia. Hysteria, psychosis, temporal lobe lesions.
3. Olfactory hallucinations. Lesions of the temporal lobe (hippocampus); partial or focal seizures of epileptic origin (uncinate seizures).

OPTICAL NERVE: II PAIR

semiography

As the examination of the optic nerve and its semiography were already studied in Section I of volume 1, here we will only remind you of its five semiographic aspects and their clinical significance:

1. Visual acuity. Remember that the Snellen chart is used to determine it. If the patient cannot read any line on the scale, the fingers of the hand are shown to him and he is asked to count them; If you can do it, you are said to have finger-counting vision. If you cannot count your fingers, but see them blurry, you are said to have lump vision. If you cannot even see your fingers dimly, you should take yourself into a dark room and, with an appropriate apparatus, project a beam of light onto the pupil; If the patient does not perceive it, he or she is said to have amaurosis, anopia or blindness.

   Decreased visual acuity may be due to:

   a) Refractive errors.

   b) Opacity of the transparent media of the eye.

   c) Retinal injuries.

   d) Injuries to the optic pathways.
2. Perinetry and campimetry. Campimetry consists of specifying the visual field, it will be explained in detail in the Ophthalmology subject, but it is important to know the alterations that this field may have, in order to interpret the report that the ophthalmologist sends to the non-specialized doctor who has requested its determination in a patient.
3. In figure 15.7 of volume 1, which illustrates the integration of the optic pathway, we see that each visual field is divided into two portions: an internal one closer to the nose and which is therefore called the nasal portion or nasal field; and an external one, which because it is closer to the temporal region is called temporal portion or field.

temporary. But it is convenient to note that the temporal portion of the visual field corresponds to the nasal portion of the retina, and that the nasal portion of the visual field corresponds to the temporal portion of the retina. If we remember what we said in Chapter 15, of the aforementioned volume, about the crossing at the level of the optic chiasm of the fibers coming from the nasal portions of each retina, we can easily understand that:

a) Total injury to an optic nerve will produce anopsia, amaurosis or blindness of the eye to which that nerve corresponds.

b) Total injury to an optic tract will produce loss of vision in the nasal portion of the visual field of the eye on the same side and loss of vision in the temporal portion of the visual field of the eye on the opposite side.

The loss of vision of half a visual field is known as hemianopia.

When the halves that have lost vision in each eye are the two right or the two left, that is, when the portion of the visual field in which vision has been lost is the nasal part of one eye and the temporal part of the other, the hemianopia is said to be homonymous.

c) When the portions of the visual field in which vision has been lost are left on one side and right on the other, as would occur in a case in which there was loss of vision in the temporal portion of the visual field of each of the eyes, hemianopia is said to be heteronymous. Thus it is understood that a lesion that sits in the optic chiasm in its anterior portion, at the angle formed by the two optic nerves, when introduced into the chiasm (as occurs in its compression by a pituitary tumor) will produce a bitemporal and, consequently, heteronymous hemianopia.

d) Lesions of the lips of the calcarine fissure will cause alterations in the upper or lower half of the visual field, as will uniform compressions of the upper or lower part of the chiasm;

We already know that the upper lip of the calcarine fissure receives the normal impulses corresponding to the upper half of the retina, and the lower lip those corresponding to the lower half of the retina. Taking into account the fibers that make up each optic stripe, we can understand that visual impulses from the upper temporal quadrant of the retina of that same region arrive at the upper lip of the calcarine fissure on each side.

side and those of the superior nasal quadrant of the retina on the opposite side; We can establish the same considerations with respect to the lower lip of said fissure. Lesions of the lips of the calcarine fissure may, therefore, produce effects in quadrants of the visual fields (the so-called quadrantanopsias), which are also produced by damage to optic radiation.

4. Color vision. The study of its alterations is the responsibility of the specialist.
5. Fundus examination. The fundus may be normal or present: atrophy of the papilla, in which case the disc appears white or pearl gray, depending on whether it is primary or secondary; edema of the papilla, swelling of the optic nerve with hyperemia and effacement of its edges and elevation. It is a fundamental sign of intracranial hypertension.

Semidiagnosis

Injuries to the visual apparatus

1. Neuritis:
   a) Retrobulbar (affects the nerve or optic pathway). It can be axial, peripheral and diffuse. The most common cause is multiple sclerosis.
   b) Optical or bulbar. It includes several forms of retinitis, as examples: simple, albuminuric, syphilitic, diabetic, hemorrhagic and hereditary.
2. Edema of the papilla. It is usually a symptom of increased intracranial pressure due to brain tumors, abscesses, hemorrhages, hypertension and many other causes.
3. Optic atrophy:
   a) Primary or simple, as in tabes and multiple sclerosis; or also, in a hereditary way.
   b) Secondary to neuritis, glaucoma or increased intracranial pressure.
4. Lens opacities. Corneal scars and atherosclerotic retinal alterations.

Visual defects

1. Scotomas, are abnormal blind spots in the visual field:
   a) Central scotoma. Loss of macular vision due to axial neuritis.
   b) Other scotomas consecutive to disseminated lesions such as hemorrhages and glaucoma.
2. Amblyopia is a defect in visual acuity, that is, decreased vision.
3. Amaurosis, is complete blindness, which can be congenital or acquired.
4. Visual field defects have already been studied; They are diagnosed by campimetry.
5. Other disorders:
   a) Hemeralopia. Day blindness; maximum vision occurs in dim light (fatigue syndrome).
   b) Nyctalopia. Night blindness, sometimes associated with a vitamin A deficiency.
   c) Color blindness. Acquired and congenital types: the latter is transmitted recessively, sometimes related to sex. Among them we have: total color blindness or achromatopsia and partial blindness: monochromatism or the ability to recognize only one of the three basic colors and dichromatism or the ability to know only two of the three basic colors.
   d) Verbal blindness: optical agnosia or inability to distinguish the objects seen. It is due to lesions of the angular gyrus.
   e) Psychic blindness. It is observed with some frequency. Experimentally it has been proven that monkeys that have suffered bilateral removal of the temporal lobes cannot recognize the objects they see.

COMMON OCULAR MOTOR, PATHETIC (TROCHLEAR) AND EXTERNAL OCULAR MOTOR NERVES (ABDUCENS): II, IV AND VI PAIRS

As we did with the exploration of these three cranial nerves, we will study their alterations together.

Semiography and semidiagnosis

Inspection of the facies allows us to realize if an eye is closed due to the drooping of the upper eyelid on that side (eyelid ptosis), which will be an index of paralysis of the levator muscle of the upper eyelid, innervated by the second pair (see Fig. 15.12, in volume 1).

The name ophthalmoplegia (from the Greek opthalmós: eye; plegée: blow) is the paralysis that affects the muscles of the eye, whether some or all of them. Depending on the location of the causes that produce them, ophthalmoplegias are classified as nuclear, supranuclear and infranuclear.

It should be noted that myasthenia gravis, exophthalmic goiter and orbital inflammations or tumors can restrict eye movements and simulate ophthalmoplegia of neurological origin.

Nuclear ophthalmoplegia

In these cases the lesion is located in the nucleus of the affected pair, in the brain stem.

In progressive nuclear ophthalmoplegia, a rare condition, symmetrical bilateral paralysis may occur, beginning with bilateral eyelid ptosis and ending in total ophthalmoplegia.

In Wernicke's superior hemorrhagic polyencephalitis, a condition seen more often in chronic alcoholics, nuclear ophthalmoplegia associated with other manifestations (delirium, disorientation in time and space, dizziness, headache and drowsiness) may occur.

Small infarcts of the brain stem can also produce nuclear palsies, which, in the case of the third nerve, can be partial or total.

Infranuclear ophthalmoplegia

In this case, these are injuries that individually affect the oculomotor nerves below their real nucleus of origin, injuries that can act at the root or along the path through the base of the skull; This is usually complete paralysis.

If it is paralysis of the third nerve, in addition to eyelid ptosis due to palsy of the levator palpebrae, when we open the eye we see that the eyeball is deviated outwards, pulled by the external rectus, and the impossibility of making eye movements up, down and in; There is also pupillary dilation and there is a lack of light reflexes (photomotor) and accommodation in that eye.

The most frequent causes are: aneurysms of the arterial circle of the brain or circle of Willys and brain tumors that cause herniation of the uncus through the tentorium cerebellum.

In paralysis of the IV nerve or trochlear, the eye is higher than normal and deviated inward; The patient cannot direct his eye downwards and outwards and diplopia appears when looking in this direction. This is why it is particularly difficult for him to descend a staircase, since he cannot look down; In addition, he habitually tilts his head forward and towards the shoulder on the healthy side.

If there is paralysis of the VI nerve or abducens, the eye deviates inwards (internal convergent strabismus) and cannot be brought outwards, resulting in diplopia when the patient tries to look in this direction.

The most frequent causes are: diabetes mellitus (polyneuropathy), intracranial hypertension of any cause (constituting a false localization sign), meningoencephalitis of the skull base and lesions of the temporal bone (petrous tip syndrome, known as Gradenigo syndrome).

Supranuclear ophthalmoplegia

In this type of ophthalmoplegia, the paralysis never affects a single ocular muscle or a single eye. On the contrary, the associated or synergistic movements of both eyes are affected. For this reason, neither strabismus nor diplopia occurs and eye movements in certain directions are abolished, but they are maintained in others, for example: inability to look to the right or left, but the possibility of ocular convergence.

Unilateral supranuclear ophthalmoplegias return to normal quickly, which is attributed to the bilaterality of supranuclear innervation.

A lesion that damages the cortical centers for conjugate gaze or interrupts their connections with the brain stem produces a conjugate paralysis of gaze to the opposite side (the patient looks at the lesion).

As stated, recovery is generally rapid.

On the contrary, when the lesion is in the brain stem (pons) and it is destructive, the patient looks towards the opposite side of the lesion. These paralysis persist longer than cortical paralysis and tend to be permanent.

In some cases the supranuclear lesion is characterized by a vertical gaze paralysis (the patient cannot raise both eyes), called Porinaud syndrome. It is most frequently caused by tumors of the pineal gland that damage the peduncular cap.

Semiography and special semidiagnosis of the intrinsic portion of the llth nerve

Pupil morphological alterations

Let's point out the most frequent ones (fig. 52.1):

Discoria. Alteration of the normal circular shape of the pupil.

Eccentric or ectopic pupil. It generally has no value as a neurological diagnosis; It may be a consequence of trauma or iritis.

Anisocoria. It consists of the inequality in size between the two pupils. It is a sign of great value in the diagnosis of cerebral vascular disease (apoplectic comas), although it can be congenital and without any diagnostic value in some people. You should always ask if mydriatics (atropin or others) have been used.

Miosis. It is the decrease in pupil size below 2 mm. Bilateral miosis is observed in the tabes dorsalis, in certain intoxications (alcohol, morphine, chloral hydrate, etc.) and in most comas, and

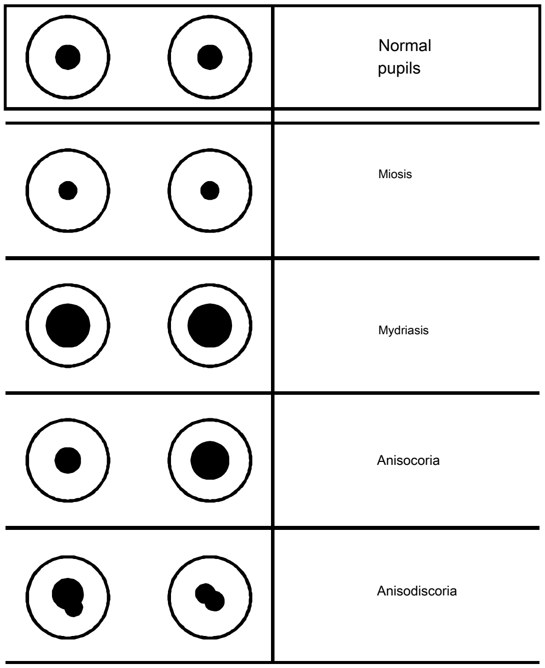


Fig. 52.1 Most frequent pupillary alterations.

reflects lower brain activity. In cases of head trauma it reveals hemorrhage from the pons with possible flooding of the IV ventricle (Alojouanine).

Mydriasis. It is the increase in the size of the pupil above 5 mm. Bilateral mydriasis is observed due to the action of some drugs or toxins (cocaine, benzene, methyl alcohol, atropine, etc.); in hypertensive encephalopathy and head trauma, in which it is intense and fixed, it carries a desperate prognosis, as it is a sign of serious brain injury, especially if pupillary reflexes are not preserved.

Alterations in pupillary reflexes

Evident pupillary hippus. It is the exaggeration of the slight contraction and dilation movements that the pupil presents, spontaneous or when the photomotor reflex is explored. Respiratory hippus is observed in subjects with labile neurovegetative systems and circulatory hippus (pupillary contraction with systole dilation with diastole) has been observed in aortic insufficiency (Landolfi sign). Provoked hippus can be seen in myotonic pupil, multiple sclerosis, Sydenham's chorea, and cerebral syphilis.

Bradychory. It is the lazy photomotor reflex, that is, the very slow contraction of the pupil under the stimulus of light; It is seen in tabes and eye diseases.

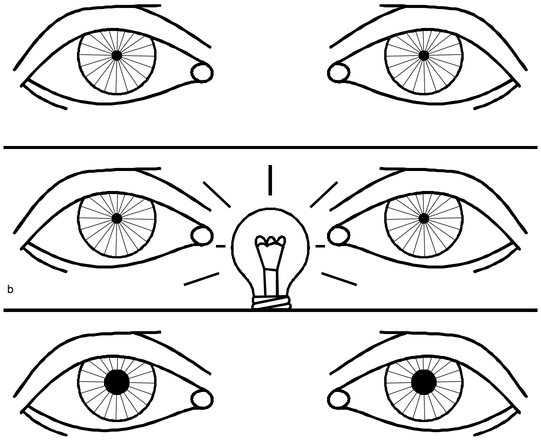
Absence of the photomotor reflex. It is the absence of contraction of the pupil under the stimulus of light; It is seen in the atrophy of the optic nerve and in the total paralysis of the third nerve together with the absence of the accommodation reflex.

Absence of the photomotor reflex and conservation of the accommodation reflex. Argyll-Robertson sign (fig. 52.2). In this case there is no pupillary response to the light stimulus; However, there is contraction of the pupil when distance accommodation is explored.

It is a very important sign in neurosyphilis. The pupil is almost always miotic.

Absence of the Consensual reflex. It is observed bilaterally when there is abolition of the photomotor reflex in both eyes. Unilaterally it depends on lesions of the optic nerve or the oculomotor nerve. If the optic nerve of one eye is affected, it will not be able to perceive light and consequently there will be no consensual reflection on the healthy side, but if we direct the light on the healthy eye, it will perceive the light stimulus and the consensual reflection will occur in the eye whose optic nerve is injured. If the oculomotor nerve of one eye is injured, the pupil will not be able to constrict even if the other eye is stimulated with light and perceives the light.

Fig. 52.2 Argyll-Robertson sign: a, miotic pupils; b, absence of light reflection; c, conservation of the accommodation reflex.



TRIGEMINAL NERVE: V PAIR

Semiography and semidiagnosis

Most common alterations:

1. Very intense pain, one of the most intense known, along the sensitive branches of the nerve. It is called trigeminal neuralgia.
2. Dissociated anesthesia of a part of the territories (already described) whose sensitivity is conducted by the trigeminal branches to the neuroaxis. This dissociated anesthesia (see dissociation of sensitivity below) consists, in this case, of the loss of thermal and painful sensitivities while preserving tactile sensitivity.
3. Paresthesias in any of the territories innervated by the V pa.
4. Herpes zoster or area of one or more branches of the trigeminal nerve. Very painful.
5. Abolition of mandibular, sneezing, palpebral, conjunctival and corneal reflexes.
6. Defective hearing due to palsy of the tensor tympani.
7. Trophic and secretory disorders. Nasal dryness, facial ulcerations, tooth loss.
8. Trisno. Very strong tonic contracture of the muscles of mastication, especially the masseter muscles, with inability to open the mouth. It is observed in tetanus, rabies and strychnine poisoning, mainly.
9. Paralysis of the chewing muscles on one side, with deviation of the mandibular towards the affected side.
10. Alternate alterations of sensitivity. To understand them we have to keep in mind that lesions of the medulla oblongata or lower part of the pons that affect the sensory pathways of the limbs and trunk (spinothalamic tracts) that have already crossed in the midline and that also affect the spinal and main trigeminal nuclei, whose fibers have not yet crossed in the midline, will produce a loss of sensitivity on the side of the face equal to that on which the lesion is located in the neuraxis and a loss of sensitivity on half of the trunk and the limbs on the opposite side equal to that on which the lesion is located in the neuraxis. This is called alternating hemianesthesia. The anesthesia that is produced is of the dissociated type like the one we described above. Alternate anesthesia is one of the members of Wallenberg syndrome, lateral softening of the medulla oblongata, due to thrombosis of the posteroinferior cerebellar artery.

FACIAL NERVE: VII PAIR

Semiography and semidiagnosis

From a semiological point of view, facial paralysis can be central or peripheral (fig. 52.3).

Central facial paralysis

It is one in which the cause that produces it is located between the motor nucleus of the facial and the area of the ascending frontal gyrus or precentral gyrus (corticonuclear motor area) where its fibers have their origin. In this case, the facial paralysis is of an inferior type, since the frontal, superficial and orbicularis oculi muscles are not affected. The paralysis of the facial is joined (on the same side as the paralyzed facial) by hemiplegia or monoplegia, since the lesion affects all the fibers of the corticonuclear bundle before crossing. Taste and salivation are not affected.

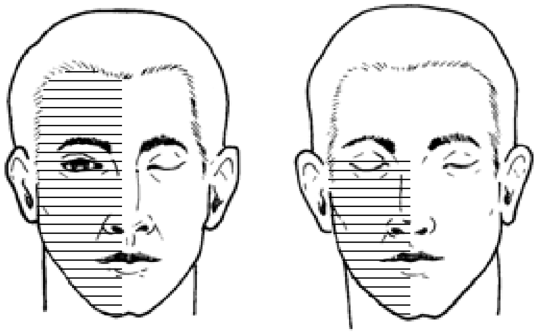


Fig. 52.3 Difference between central and peripheral facial paralysis: a, peripheral facial paralysis: the eye remains open (lagophthalmia) because the upper facial is injured in the paralysis; b, central facial paralysis: occlusion of the eye is possible because the upper facial is not injured.

This type of paralysis is also called supranuclear. It is most frequently caused by tumors and brain vascular accidents.

Peripheral facial paralysis

It can be nuclear or infranuclear depending on where the lesion is located.

Nuclear facial paralysis

Paralysis of all the muscles of the face on the same side as the one on which the injury is located is observed. In addition, there will be hemiplegia on the side opposite to that on which the injury occurs. The VI nerve may also be paralyzed, on the same side as the injury.

Example: an injury to the right side of the crown of the annular bridge will damage the nucleus of the VII pair and perhaps the VI on the same right side, and since the fibers that come out of them have already crossed, the paralysis of the face will be on the right side and in the abducens muscle of the eye on the right side; but the corticonuclear pathway (at that height) has not yet been crossed, the lesion will damage the fibers there that were destined for the extremities on the left side. This produces a particular type of hemiplegia, which is called alternating hemiplegia.

Infranuclear facial palsy

Also called total facial paralysis, Bell type palsy or upper and lower type paralysis.

The facies and other characters will depend on the site where the injury occurs:

1. Injury at the outlet or beyond the stylomastoid foramen.
   Paralysis affects the entire side of the face where the injury that causes it is located. The forehead of the paralyzed side appears smooth, without wrinkles, contrasting with the healthy side. The eyebrow on the paralyzed side appears drooping, lower than that of the healthy side, with the impossibility of voluntarily raising it or frowning on that side. The eye on that side is more open than the one on the healthy side, due to paralysis of the orbicularis oculi, which is known as lagophthalmia or lagophthalmos. If you try to close your eye, the eyeball moves upward and outward (Charles Bell sign). The patient cannot completely occlude the diseased eye. There is ephora (tearing) and paralysis of the Horner muscle if the eye is not protected. The cheek of the paralyzed side is flaccid and projects more markedly during expiration than that of the healthy side. The lip corner is slightly deviated towards the healthy side. There is difficulty swallowing due to food accumulating between the cheeks and gums and for the same reason there is salivation or drooling. The patient cannot whistle. The paralysis is of the flaccid type (see "Motility" in the previous chapter).

2. Injury to the facial nerve canal or fallopian aqueduct that damages the chorda tympani, that is, above the chorda tympani, but within the aqueduct. He will have all the signs mentioned, plus the loss of the sense of taste in the anterior two-thirds of the tongue and reduced salivation on the paralyzed side.

3. Injury to the facial nerve canal or fallopian aqueduct, beyond the emergence of the branch that innervates the stapes muscle. All the symptoms indicated in 1 and 2, plus hyperacusis (exaggerated and painful hearing).

4. Injury to the facial nerve canal or fallopian aqueduct, even higher, at the level of the geniculate ganglion.
   Everything mentioned in 1, 2 and 3, plus pain inside and behind the ear. When accompanied by vesicles located in the external canal and in the pinna of the ear, it constitutes Ramsay-Hunt syndrome or geniculate ganglion syndrome of viral etiology (herpes zoster).

5. Injury to the internal auditory canal. Everything indicated in 1, 2, 3 and 4 plus deafness or decreased hearing on the affected side, since pair VIII is also found within this canal.

6. Injury between the entrance (endocranial orifice) of the internal auditory canal and the emergence of the facial canal in the pons (injury in its intracranial path). Everything indicated in 1, 2, 3, 4 and 5, plus symptoms belonging to other neighboring nerves such as the V pair and sometimes VI, XI and XII pairs. This is the site where tumors of the cerebellopontine angle occur, caused by an acoustic neuroma.

   The most common cause of peripheral facial paralysis is the so-called frigore paralysis, supposedly caused by viruses. Other causes may be intrapetrous inflammatory processes, parotid tumors, and traumatic or surgical sections of the nerve.

Semiographic alterations of the sensory portion

Many times before the examination the patient informs us that he "does not taste" what he ingests; that what you eat or drink tastes the same. Other patients complain of a bland taste.

The main alterations found are:

1. Ageusia. Loss of sense of taste.
2. Hypogeusia. Decreased sense of taste.
3. Parageusia. Confusion or perversion of flavors.

The alterations corresponding to the sensory portion have been studied together with those of the motor portion.

STATOACUSTIC NERVE: VIII PAIR

COCHLEAR NERVE

Semiography and semidiagnosis

Hearing disorders of neurological origin are the following:

1. Ringing in the ears (tinnitus). It may subjectively have a different tone or character, and its intensity may be constant or variable. It is caused by lesions of the middle ear (otosclerosis), the labyrinth or the cochlear nerve. When it comes to injuries to this last nerve, it always precedes hearing loss or deafness.

   It is so constant in lesions that affect the cochlear nerve that it can be said that if a patient does not have or has not had tinnitus, he or she should not have deafness caused by injury to the VIII nerve. In these cases, tinnitus is often accompanied by symptoms related to neighboring structures (vestibular nerve, VII nerve, stem, cerebellum). The most common cause is neuromas of the VIII nerve.

2. Hearing loss or deafness. That produced by lesions of the cochlear nerve (perceptual or nervous deafness) is characterized

   a) Be preceded by tinnitus.

   b) Decrease or loss of hearing conducted through the bones of the skull.

   c) Partial loss of high-pitched sounds.

   Partial deafness due to conduction disorders (produced by middle ear lesions) is characterized, on the contrary, by intensification and prolongation of bone hearing and partial loss of the perception of low sounds.

   Hearing loss or deafness can also be caused by lesions of the brain stem (rare) and, even more rarely, by lesions of the cerebral cortex. In these cases, if the lesion is unilateral, the hearing loss is temporary, and is definitive only in cases of bilateral cortical lesions. Even in this type of lesion, as noted, the patient may be able to hear noises and perceive differences in tone (representation in subcortical structures).

3. Paracusia (from the Greek, para: next to: akozucin: hear).

   Hearing is better in noise than in silence (generally due to middle ear injuries).

VESTIBULAR NERVE OR BRANCH

Semiogenesis and semiography

1. Vertigo. The word vertigo comes from the Latin vertere, which means to turn. Vertigo is characterized by a distressing sensation of displacement and rotation. Only frank and definitely rotating sensations will be admitted as vertigo, whether the patient reports that things are spinning around him (objective vertigo) or if he is the one spinning (subjective vertigo). Other types of instability, poorly defined, such as dizziness and fainting, should not be accepted as vertigo. Another less common type of vertigo includes the sensation of sinking or moving violently and suddenly in space.

   Vertigo can occur isolated or accompanied by vagal manifestations (vegetative component): paleness, sweating, vomiting, diarrhea, bradycardia and a feeling of anxiety, which, together with vertigo, are particularly unpleasant for the patient.

2. Nystagmus. It is a rhythmic movement of the eyeballs with a slow phase and a fast phase in the opposite direction to the previous one, and which is the one that is collected to indicate its direction: horizontal (to the right or left), vertical (up or down) and rotary or mixed. Nystagmus seems to represent a disorder of the complex system that keeps the eyes in constant relationship with the environment. It includes the retinas, the vestibule of the inner ear, the vestibular pathways, the cerebellum and the connections with the oculomotor pairs; all controlled and modulated by the cerebral cortex.

3. Balance disturbances. They are expressed by gait alterations, the positive Barany index deviation test and the Romberg sign, also positive.

   Romberg's sign occurs in vestibular syndrome and posterior cord syndrome. It is explained by admitting that to maintain balance, man has three sources of information: 1 vision, the labyrinth and the vestibular pathways, and deep sensitivity. The balance is maintained if two of these information channels are undamaged.

   As said in the study of ataxia (Chapter 51), during the Romberg maneuver, the patient must close his eyes. This eliminates one of the three sources, so if there is a vestibular pathology or a lesion of the posterior cord (through which deep sensitivity is conducted) the patient will not be able to maintain balance.

Semidiagnosis

1. Vertigo. It is almost always of labyrinthine origin (peripheral vertigo). It is accompanied by vagal manifestations with some frequency. It is a symptom for which it is often consulted. Its causes are varied; sometimes occurs only with changes in head position

(benign position vertigo) and is seen in: acute labyrinthitis (benign process of possible viral origin), Meniére's syndrome and toxicity due to streptomycin, kanamycin or gentamicin. Vertigo of central origin is rare, and occurs in some types of rare epileptic seizures, in seizures of ischemia of the vertebrobasilar territory or in cerebellar hemorrhages or multiple sclerosis. It should be noted that isolated vertigo is not an expression of cerebral ischemia. This vertigo is not accompanied by the vagal manifestations described.

2. Nystagmus. It can be of three types:

a) Nystagmus of ocular origin. It can be physiological, when there is continuous movement before the eyes, for example, when looking out the window of a moving train.

Pathological ocular nystagmus occurs when there is a significant visual defect from birth, since macular fixation of vision and the ability to maintain it develops in the first months of life. It is due to cranial injuries, congenital cataract, albinism. Ocular nystagmus is rapid and pendulous, without fast and slow phase, more often horizontal, and persists throughout life.

b) Nystagmus of vestibular origin. It is almost always horizontal or rotating. It may be due to multiple processes that were pointed out when referring to vertigo of vestibular labyrinthine origin. It is often associated with vertigo, which

It can never be vertical. Vertical nystagmus is always central (neurological).

c) Nystagmus originating in the central nervous system.

It almost always occurs due to injuries that affect the region of the fourth ventricle and more rarely in cerebellar conditions. It can be: horizontal, vertical, rotating or even different in both eyes.

It is almost never associated with vertigo. It occurs in multiple sclerosis, in diseases that affect the central portions of the cerebellum, in transient cerebral ischemia of the vertebrobasilar territory; in toxicity due to diphenylhydantoin and carbamazepine, etc.

GLOSSOPHARYNGEAL, PNEUMOGASTRIC (VAGUS) AND ACCESSORY NERVES: IX, X AND XI PAIRS

GLOSSOPHARYNGEAL NERVE: IX PAIR

semiography

1. Exploration of taste in the posterior third of the tongue. When this exploration is performed by applying co- galvanic current of 0.25 6 0.50 mA (milliampere) on the tongue and there is no perception of acid taste (ageusia), this is highly suggestive of injury to the IX nerve.
2. Pharyngeal reflex. Lesions of the IX nerve are characterized by loss of the pharyngeal reflex, since this cranial nerve offers the centripetal (sensory) pathway for it; but it is only significant when the areflexia is unilateral, since the bilateral absence of this reflex is normally not rare.
3. Carotid sinus reflex. It is actually used as a therapeutic and not a diagnostic method.
4. Vernet phenomenon. The contraction of the posterior wall of the pharynx, when the patient says "a", does not occur if the IX nerve is injured.

Semidiagnosis

Isolated paralysis of the IX nerve is rare. It is associated with that of the

Neuralgia of the IX nerve is one of the few conditions of this nerve individually. Patients experience lancinating pains in the lateral wall of the pharynx and tonsillar region that radiate to the Eustachian tube or auditory tuba, the eardrum, and the external auditory canal.

PNEUMOGASTRIC NERVE (VAGUUS): X PAIR

Semiography and semidiagnosis

1. Paralysis of the soft palate. Unilateral has already been described. Bilateral involves bilateral vagus paralysis, which is quickly fatal.
2. Voice alterations. They can be:

a) Aphonia. It is the loss of the voice due to paralysis of the vocal cords or hysteria.

b) Bitonal voice. It is characterized by the spontaneous change in tone of the voice while speaking; It goes uninterruptedly from a low tone to a high tone and vice versa. It is generally due to paralysis or paresis of a vocal cord.

c) Paralysis of a vocal cord. Using the laryngoscope, it is observed that one of the vocal cords lacks movement. It is one of the causes of bitonal voice. It can be caused by nuclear or peripheral injury to the vagus nerve on the same side, almost always due to tumor or toxic vascular problems.

In these cases, the symptoms dependent on the interruption of the sensory pathways will be added to the paralysis of the vocal cord: anesthesia of the pharynx on the affected side and on the same side of the larynx in the case of peripheral paralysis and loss of sensitivity to pain and temperature on the opposite side of the lesion, in addition to the above, in the case of nuclear seat palsy.

3. Oculocardiac reflex. It has already been explained in previous paragraphs.

ACCESSORY NERVE: XI PAIR

Semiography and semidiagnosis

Paralysis caused by injury to the XI nerve can be:

1. Unilateral. Due to peripheral injuries. It is manifested by:
   - a) Impossibility of head rotation to the side healthy.
   - b) Atrophy of the sternocleidomastoid, with:
     - Degeneration reaction.
     - Inability to elevate the shoulder on the diseased side mo.
     - Dropped shoulder on the affected side.
     - Downward displacement of the scapula.
     - Depression of the shoulder contour due to trapezius atrophy.
2. Bilateral. Due to nuclear or peripheral injury:
   - a) Difficulty in rotating the head or lifting the chin (sternocleidomastoid).
   - b) Falling of the head forward: square shoulders due to atrophy of the trapezius.
3. Central. It produces similar movement limitations, but does not cause muscle atrophy or a degeneration reaction. The muscles are spastic and, if the lesion is unilateral, there may be torticollis.

MAJOR HYPOGLOSSAL NERVE: XII PAIR

Semiograph

1. Trophism and fasciculations. When there is atrophy on one side, the paralyzed side appears more depressed vertically, more flattened than the normal one. When there is atrophy of half of the tongue, the atrophied side that corresponds to the same side as the nerve lesion presents the characteristics described. There may be fasciculations.
2. Central or deviated position of the tongue. The side towards which the tip deviates is the side on which the lesion is located. Remember to rule out false deviations, when there is facial paralysis or when teeth are missing.
3. Segmental muscle strength of the tongue, seeking its decrease.

Semidiagnosis

Central (supranuclear) palsies of the XII nerve practically do not produce clinical alterations, except for the deviation of the tip of the tongue towards the paralyzed side (contralateral lingual palsy).

Nuclear ones also cause atrophy of the tongue on the affected side, with fasciculations. They are observed in bulbar hemorrhage, tumors, multiple sclerosis, poliomyelitis, etc.

The peripheral ones give rise to ipsilateral paralysis of the tongue and atrophy on that side. When the tongue is protruded, it is deflected towards the side of the lesion by the action of the genioglossus on the opposite side.

It is seen in fractures of the base of the skull, in upper cervical dislocations and in poisoning by alcohol, arsenic, lead (lead poisoning), etc.

NERVE SYSTEM0. COMPLEMENTARY EXPLORATIONS

53

The semiology of the nervous system obtained through a complete clinical examination constitutes the cornerstone for the approach to patients, the fundamental source for the diagnosis. This cannot be replaced by any other exploration that has emerged as a particular expression of the great scientific-technical development of our time.

Consequently, the use of so-called complementary techniques for the study of neurological patients and the assessment of their results must be framed in two fundamental principles:

1. The data from the interrogation and physical examination should guide your indication.
2. The information they provide does not replace clinical information, which is why they must be integrated with it for their proper evaluation.

3. Used correctly, they often offer extremely precise data, and like the clinical part, they cannot be substituted or ignored for the study of patients.

We will make a brief description of the main complementary tests commonly used in Neurology, insisting on their indications and the data they can provide to the medical diagnosis.

The most commonly used complementary examinations in the nervous system are:

Lumbar puncture and study of cerebrospinal fluid (CSF) or cerebrospinal
Imaging examinations.
Exploration of the central nervous system using radioisotopes.
Electroencephalography.
Evoked potentials.
Electromyography and electrical stimulation of muscles and peripheral nerves.
Biopsies.

LUMBAR PUNCTURE AND STUDY OF THE CEPHALORSPINAL FLUID (CSF) OR CEREBROSPINAL

This test is very useful but must be used with good judgment, since it is not without risks. As this is a relatively bloody test, care must be taken to obtain the greatest precision in its performance and interpretation, so that it does not have to be repeated unnecessarily.

Lumbar puncture is performed by inserting a needle into the cul-de-sac of the spinal canal, to:

1. Measure CSF pressure.
2. Observe the macroscopic appearance of the CSF.
3. Subject the CSF to various laboratory tests.
4. Perform, if necessary, tests that determine whether there is blockage of the subarachnoid space.

ANATOMY OF THE SYSTEM THAT HOUSES THE CSF

The cerebrospinal fluid (CSF) or cerebrospinal fluid is an organic, transparent humor with a watery and slightly alkaline appearance, with a density approximately that of water that is housed in the ventricular system of the central nervous system (CNS) and in the subarachnoid space (fig. 53.1).

It originates mainly at the level of the choroid plexuses located on the roof of the third and fourth ventricles and on the floor of the body and lower horns of the lateral ventricles.

The choroid plexuses of the lateral ventricles are the main producers of CSF (95%), from where it passes through the interventricular foramina (foramina of Monro) to the third ventricle; from there, to the aqueduct of the brain (Sylvian aqueduct) and later to the fourth ventricle, from where it drains into the subarachnoid space at the level of the cerebellomedullary cistern (magna), through the lateral openings (foramina of Luschka Magendie), from where it diffuses over the brain and spinal cord until reaching the reabsorption points.

In addition to the choroid plexuses, the ependyma participates in the production of fluid.

By experimental methods it has been calculated that the choroid plexuses produce approximately 250 mL daily. Under normal conditions the total volume of liquid is 150 mL, so it is constantly renewed several times a day.

The point of maximum resorption is found at the level of the arachnoid villi or granulations, which are protrusions of the arachnoids towards the venous sinuses of the dura mater, mainly in the superior sagittal sinus. The speed of CSF reabsorption depends on its pressure, since the arachnoid villi act as valves in only one direction. Some of the fluid is also reabsorbed in the ependymal tunic of the ventricles and through the capillaries of the pia mater.

FUNCTIONS OF CSF

1. The CSF probably acts as a kind of cushion for the brain, preventing or reducing the transmission of shocks or shocks that could act on the brain and spinal cord.
2. Its metabolic role is problematic. It is possible that the CSF delivers nutritional materials to the nervous centers and carries away the metabolites.
3. Changes in intracranial volume are sometimes compensated by CSF production. This is clearly seen in destructive or postoperative injuries.
4. It would control the cellular and humoral immunity of the central nervous system, since in the course of some immunopathological processes (type of plaque sclerosis, subacute sclerosing leukoencephalitis) certain products that have been designated as immunological metastases would be found in the CSF.

LUMBAR PUNCTURE TECHNIQUE

A prior explanation must be made to the patient and his family of the need for him to undergo the test with as little apprehension as possible.

Sterilized material is required: trocar about 8-10 cm long (18-21 gauge), several test tubes, swabs, ordinary forceps and surgical gloves.

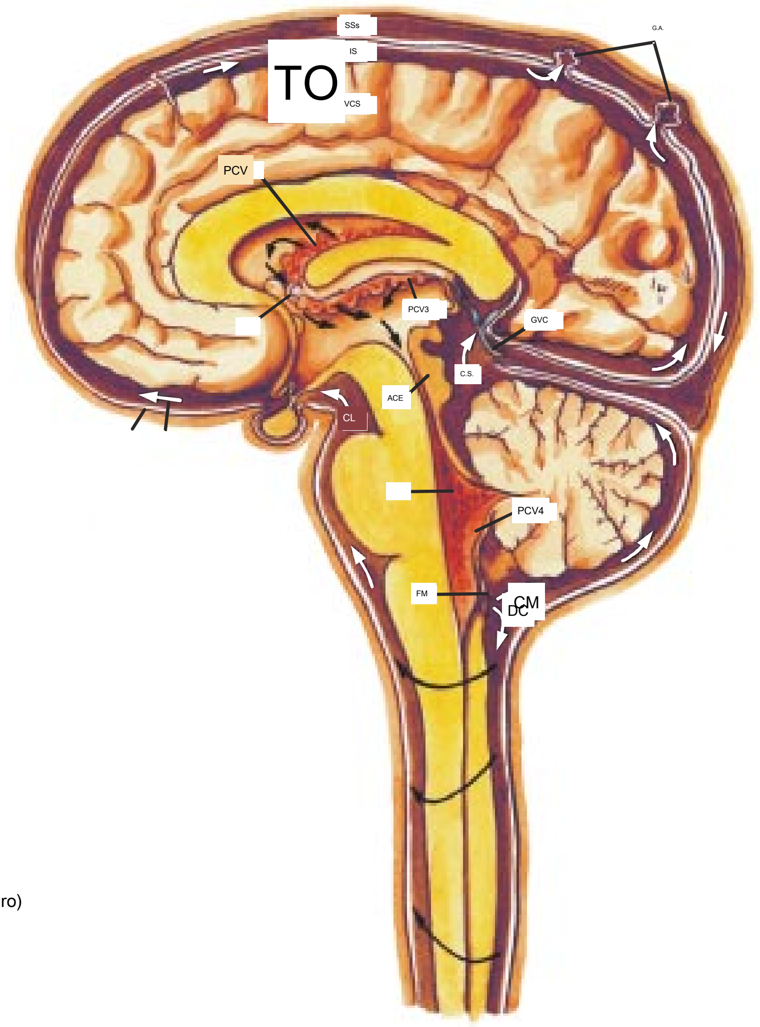
The patient must remain in lateral decubitus, lying on a rigid surface (edge of the bed); The thighs should be flexed as much as possible on the belly (knees towards the beard) and the trunk on the chest. An assistant or nurse should prevent the patient from leaving this position.

Local anesthesia of the skin and underlying tissues is performed using a 26 or 27 gauge needle.

The back must be uncovered from the lower part of the dorsal column to the very beginning of the gluteal region, to proceed to sterilize with alcohol or merthiolate, the area where the puncture will be performed: space between L3 and L4 (although a space can be made higher or a space lower). The reference point is the iliac crest; The interspinous space that remains at its level is the chosen site. This space can be determined quite accurately by drawing an imaginary line that joins both iliac crests. With gloved hands, the space is well specified with one finger and with the other hand the trocar is introduced in a perpendicular plane (somewhat inclined upwards) and without any lateral deviation, but strictly in the anteroposterior direction.

Normally, the doctor feels the sensation of the trocar as it passes the ligamentum flavum and passes through the dura mater. The mandrel is then removed from the trocar, through which the CSF should immediately flow, drop by drop.

If this does not happen, the trocar can be moved a few millimeters backwards. If bone is found, it should be removed almost until it is extracted and directed again. If it deviates



A: arachnoid
GA: arachnoid granulation
AS: Silvio aqueduct
CI: cerebellomedullary cistern
CCM: cerebromedullary cistern
PCV4: Choroid plexO of 4t0. ventricle
PCV3: 3rd choroid plexO. ventricle
PCVL: Choroid plexO of the lateral ventricle
CS: upper tank
D: dura mater
FL: Luschka foramen
FM: Magendie foramen
GVC: great cerebral vein
FI: interventricular foramen (foramen of Monro)
EN: subarachnoid space
SVC: superior cerebral vein
SSS: superior sagittal sinus

Fig. 53.1 Origin and circulation of cerebrospinal fluid.

Laterally, the patient may complain of sudden, shooting pain in one leg, indicating that a posterior root has been punctured and that the trocar should be redirected to the midline. If blood comes out and then clears, it means that we are inside the canal, but that the puncture was traumatic; If only blood comes out, the trocar should be withdrawn a few millimeters back and try again. At the end of the lumbar puncture, the trocar must be removed quickly and the patient should rest in bed for at least 24 hours.

The appearance of CSF is usually clear as rock water. To measure the pressure, the Claude manometer is used. The normal initial pressure should be between 7 and 18 cm of water. The usual pressure is 8-14 cm of water.

If the CSF is hemorrhagic, the test should be done by collecting it in three separate tubes, each with 2-3 mL. If the color is the same in all three, it is not a traumatic puncture, but rather blood in the CSF (subarachnoid hemorrhage, cerebral hemorrhage with ventricular flooding).

If the initial pressure is above 18 cm of water, we must ensure that no external factor intervenes, such as exaggerated flexion of the neck or legs, a pillow compressing the neck or the patient contracted, breathing shallowly. In this case we must eliminate the obstacle and repeat the observation of the CSF pressure. Figures above 20 cm are definitely hypertensive. During observation, an up and down movement of approximately 1 mm can be seen in relation to respiratory movements, which indicates that the needle is not obstructed.

QUECKENSTEDT-STOOKEY TEST

Compression of the jugular veins produces an increase in pressure. The compression is maintained for 10 s and is then abruptly removed. After 10 seconds, the pressure should return to the initial figure. If the pressure does not rise above 20 cm of water during compression, or if after 10 s of cessation of compression it remains at a higher figure than the initial one, it can be diagnosed.

a blockage (total or partial) of the spinal subarachnoid space.

CSF EXAMINATION

Normal characteristics of CSF

Appearance: like rock water
Pressure: 7-18 cm of water
Proteins: 15-45 mg/dL (in lumbar region)
Protein electrophoresis:

- Prealbumin: 2-6%
- Albumin: 44-62%
- Alpha 1 globulin: 4-8%
- Alpha 2 globulin: 5-11%
- Betaglobulin: 13-26%
- Gammaglobulin: 6-13%

Glucose: 50-75 mg/dL (2.8-4.2 mmol/L)
Chlorides: 120-130 mEq/L (20 mEq higher than serum)
Wasserman reaction: negative
Colloidal gold curve: 000000000 - 0001222111
Direct examination and bacteriological culture: no germs
Cells: 0-5 mononuclear cells

Amount of CSF required for each test

Proteins: 1.5-2 mL
Protein electrophoresis: 10 mL
Glucose and chlorides: 3 mL
Wasserman reaction: 2 mL
Colloidal gold: 0.5 mL
Culture and direct examination: 2 mL (for tubercle bacillus, 3-5 mL)
Cell count: 0.5 mL

Nowadays, with the use of micromethods, the amount of CSF required is minimal.

INDICATIONS FOR LUMBAR PUNCTURE

It is indicated in cases of meningoencephalitis, cerebral vascular accidents, paraplegia and to perform other tests (pneumoencephalogram).

CONTRAINDICATIONS OF LUMBAR PUNCTURE

In intracranial hypertension. In these cases it can only be performed in coordination and under the supervision of the neurosurgeon, in a center prepared for neurosurgical interventions (danger of jamming of the brain stem in the tentorium cerebellum or of the cerebellum bulb in the foramen magnum).

INFORMATION YOU CAN PROVIDE

1. In the event that there is a blockage to CSF circulation in the lower part of the spine (spinal tumors), In addition to the Queckenstedt-Stoockey test being positive, the liquid is xanthochromic (xanthochromic: yellow) and presents a marked increase in proteins, which is why it can clot.
2. Albumino-cytological dissociation is typical in Guillain-Barré syndrome: normal cells and a marked increase in proteins.
3. Marked decrease in glucose in purulent bacterial meningoencephalitis.
4. Wasserman reaction and positive colloidal gold curve in cases of syphilis of the central nervous system.
5. Red fluid in all three tubes in pure subarachnoid hemorrhage and in subarachnoid hemorrhage secondary to cerebral hemorrhage. If allowed to settle, the supernatant is xanthochromic. A few days after bleeding, the CSF is xanthochromic with elevated albumins.
6. Meningoencephalitis. In the bacterial case, the study of CSF is essential. The macroscopic appearance (turbid or clearly purulent) allows early decisions to be made about treatment, without having to wait for bacteriological studies; The cell count explains the appearance: 400 or more white cells, with more than 70% polymorphonuclear. A Gram stain can be useful to guide the causative germ, but the true diagnosis of this can only be made with the culture.

In viral meningoencephalitis the CSF is clear (unless the number of cells is very high in which case it may be somewhat cloudy) and the cell count is high and with a predominance of lymphocytes.

In tuberculous meningoencephalitis the fluid has the classic appearance of rice water with some fibrin meshes. The cell count is 50-200 cells, with a predominance of lymphocytes. Glucose and chlorides are low, a fact that when found has diagnostic value, but only the isolation of the Koch bacillus by culture or inoculation to the curiel allows us to ensure.

rar it.

IMAGING EXAMINATIONS

These exams will be studied in more detail in the corresponding subject. Here we will analyze the indications and the data they can provide:

1. Simple skull x-ray.
2. Contrasted radiological examinations:
    a) Pneumoencephalogram.
    b) Cerebral angiography.
    c) Myelography.
3. Echoencephalography or brain ultrasound.

4. Computed axial tomography (CAT).
5. Nuclear magnetic resonance (MRI).

SIMPLE SKULL X-RAY

A front view and a side view will always be requested.
In the frontal view, the orbits, sphenoidal wings and indentations, and the frontal and maxillary sinuses are better seen. The lateral view allows you to see the sella turcica, the floor of the anterior fossa, and the mastoids. If you want to see the rocks, the posterior fossa and the foramen magnum, you can do special views (Stenvers, Towne, Hirtz, etc.).

Indications

Cranial trauma, intracranial hypertension, suspected brain tumor, suspected intracranial expansive masses, congenital anomalies and inflammatory bone processes.

Information you can provide

1. Fracture lines.
2. Separation of cranial sutures in children with intracranial hypertension.
3. Erosion of the sella and clinoid processes, in intracranial hypertension.
4. Deviation of the pineal gland from the midline (when it is calcified by any expansion).
5. Calcifications (infectious tumors or parathyroid disorders). (Calcification of the falx cerebri and choroid plexuses may be normal, as may calcification of the pineal gland.) 6. Erosion of the auditory canal (neurinoma of the VIII nerve).
7. Increase in diameters and effacement of the sella turcica (pituitary tumors).
8. Osteolysis, osteosclerosis or localized bone proliferation (tumors).
9. Congenital anomalies (platybasis or basilar impression).

PNEUMOENCEPHALOGRAM

Injection of air into the subarachnoid space through a lumbar puncture. Air fills the ventricles, allowing their shape, size and position to be observed. It is an exam that is performed only in specialized services.

Indications

Suspected intracranial expansive mass, or other types of lesions without clear localization signs.

Information you can provide

1. Ventricular dilation (hydrocephalus, cerebral atrophy).
2. Displacements (expansive mass).
3. Amputation of an extension, localized deformity (expanding mass).

Risks

Nailing in cases of intracranial hypertension.

CEREBRAL ANGIOGRAPHY

It consists of the visualization of the arteries and veins of the brain and spinal cord, through their opacification with an iodinated contrast. Except for the possibility of an anaphylactic reaction, tolerance to current non-ionic contrast agents is very good.
Angiography is absolutely contraindicated in patients with known allergies to iodinated contrast and should be performed with extreme caution, and with all resuscitative measures at hand, in patients with other types of allergic reactions. The patient must be fasting and mild sedation is advisable.

In children and people who do not cooperate, general anesthesia is essential. Angiography carries a mortality risk of less than 1 per 1,000 and a morbidity risk of less than 2%. The most common complications are: hematomas at the puncture site, spasms or thrombosis of the punctured artery (usually the femoral artery) and cerebral embolisms; Patients should be closely monitored in the hours following angiography in anticipation of these complications. It is rare for a local hematoma to require evacuation; femoral thromboses with acute leg ischemia require immediate consultation with a vascular surgeon and, eventually, thrombectomy; Cerebral embolisms can be treated with vasodilators and anticoagulation with heparin, if they are identified in the first minutes. Morbidity is directly linked to the surgeon's experience, the number of arteries explored and, therefore, the duration of the examination.

Modern digital angiography equipment allows for faster examination, with less contrast and with automatic subtraction images on the spot.
However, its resolution is lower and the definition of small-caliber arteries and veins is worse than with conventional fine-focus equipment.

Technique

The usual technique for angiography is femoral puncture and selective catheterization. For cerebral arteriography, the carotid and vertebral arteries are characterized; Depending on the indication, it may be sufficient to inject only one artery, as would be the case of a hemispheric expansive process;
but frequently several trunks have to be injected, even all four, and sometimes also the external carotids and other extracranial branches. These cases correspond to patients with arterial stenosis and complex collateral circulation, arterial or arteriovenous malformations, meningiomas, dural or carotid cavernous fistulas,

gliomic tumors, etc., where very precise knowledge of the vascularization of the lesion is necessary to make a therapeutic decision, whether endovascular or combined surgery.

Spinal arteriography is technically very complicated, because the afferents to the spinal cord are multiple through radiculomedullary arteries that arise from the vertebral and intercostal arteries on both sides and therefore require catheterization of multiple pedicles. The indications for spinal angiography are very selective in the case of suspected intra- or extradural vascular malformations and some intramedullary tumors (hemangiomas).

Information you can provide

1. Visualize the aneurysms or angiomas in relation to the arteries on which they depend.
2. Displacements of large arteries (tumor).
3. Localized abnormal vascularization (tumor).
4. Cortical avascular zone (subdural or epidural hematoma).
5. Arterial atheromatosis.

Risks

Among the risks we can have cervical bleeding due to hematoma at the puncture site, seizures and temporary or permanent hemiplegia.

MYELOGRAPHY

It consists of the visualization of the spinal subarachnoid space, by means of a radiopaque iodinated contrast that is introduced by lumbar puncture. The current non-ionic contrasts are very little irritating and can be used throughout the entire length of the spinal canal and even in the cranial cavity; With the old iodinated contrasts, their passage into the cranial cavity greatly increased the incidence of undesirable effects such as headaches and vomiting, and above all, epileptic seizures and confusional states.

The main indications for which the clinician may require a myelography are suspicions of cervical spondylotic compression, syringomyelia, expansive processes at all levels, acute traumatic paraplegia or due to neoplastic compression or hematomielia, and radicular syndromes in the legs, either with involvement of one root or several (horsetail). In all these cases, magnetic resonance imaging (MRI), alone or combined with computed axial tomography (CAT), is clearly superior to myelography. If access to MRI is difficult, myelography may be indicated in emergency situations (acute spinal compressions). It can also resolve doubts in some cervical or lumbar root compressions due to very lateral disc herniations that are of difficult diagnosis in other imaging tests. In the diagnosis of arteriovenous malformations, myelography is a useful intermediate step between MRI and arteriography; With the patient in the supine position, the large drainage veins are very well observed as negative, serpentine tubular images within the iodinated contrast. A completely normal myelography makes the existence of a dural arteriovenous fistula in the spine unlikely.

When in myelography the transit of iodinated contrast through the lumbar route is completely stopped by an expansive process that blocks the subarachnoid space, it is essential, if a surgical treatment is considered, to delimit the upper level of the blockage by injecting contrast through a suboccipital puncture.
Otherwise there is a risk that the laminectomy will not focus on the exact level of the tumor.

Classic myelography, in which contrast is observed in the magnifier and anteroposterior and lateral radiographs are taken, can be completed with CT immediately or after a few minutes. Delayed CT (at 60-120 minutes) allows us to check how syringomyelic or arachnoid cysts are filled with contrast, but it is also a technique that is progressively falling into disuse after the arrival of MRI.

ECOENCEPHALOGRAPHY OR BRAIN ULTRASOUND

It was introduced in 1956. It is based on the principle of detecting faults or reflections (echoes) of an ultrasonic current passing through the skull. It allows recognition of lateral displacements of the midline supratentorial structures, as well as ventricular enlargements, which is why it is useful in supratentorial lesions that occupy space (hematomas, tumors, abscesses, hemorrhages). The deviation of the structures from the midline can be determined with an accuracy of 1-2 mm. A deviation of 3 mm or more is considered abnormal.

Imaging the brain and its ventricles using ultrasound has become the standard examination in the case of premature babies, neonates and infants due to its accessibility, safety and high resolution, taking advantage of the window that the open frontal fontanelle offers to ultrasound. Hemorrhages, calcifications, postencephalitic cysts, postmeningitis and hydrocephalus can be detected, which allow the diagnosis and monitoring of most of the serious brain processes of this stage of life.

COMPUTERIZED AXIAL TOMOGRAPHY (CT)

This method constituted a revolution within medicine and particularly radiology; It was introduced into the clinic around 1972.

It uses a very crowded (non-flare) x-ray beam, which scans the patient's head, for example, in several readings or slices. This x-ray beam passes in a straight line through the head and will hit a highly sensitive scintillation detector. Both the x-ray beam and the detector rotate around the patient's head, so that multiple slices are made.

Each rotation is one degree. 180 records or scans are produced, allowing 28,000 cuts or readings to be made. These readings are processed in a minicomputer that calculates the absorption values of the material in the cut. From these calculations, a three-dimensional image (matrix) emerges that shows the different densities of the soft tissues explored with

This system is 100 times more sensitive than conventional radiological examination. The irradiation dose is approximately equivalent to that of standard radiological examination.

The method is free of risks and discomfort for the patient; When using iodinated contrast, one should beware of allergic reactions.

In any case, CT is today the basic brain imaging technique in most hospitals, especially for emergency cases (trauma, heart attacks, cerebral hemorrhages) and for cases of suspected supratentorial expansive processes.

CT offers precise information on the disc, joint and vertebral pathology of the spine, especially the lumbar spine. It is not as useful in the cervical spine where, since there is no epidural fat, there is not enough natural contrast to detect herniated discs. In this case, it is advisable to combine CT with myelography.

Information that provides

1. The cerebral ventricles and the subarachnoid space are seen, so that cerebral atrophy or any alteration in the position or shape of the ventricles can be detected.

2. The extravasated blood has much greater density than the nervous substance in the method, so parenchymal hemorrhages can be detected, even small ones, as well as blood in the ventricles. Non-hemorrhagic cerebral infarctions can also be detected.

3. Brain tumors of any nature are easily detected, differentiating them from brain abscesses. Subdural hematomas are difficult to see, particularly when they are bilateral and do not produce ventricular displacement. Intracranial aneurysms and arteriovenous malformations do not occur unless they contain calcium or clotted blood.

This method has displaced brain exploration with radioisotopes, echoencephalography, electroencephalography and conventional radiological studies (with the exception of cerebral angiography) in the study of intracranial tumors and cerebral vascular accidents; However, the equipment is very expensive and requires special training of the radiologist.

NUCLEAR MAGNETIC RESONANCE (NMR)

This study technique has had a rapid development and has replaced CT as the examination of choice in most neurological disorders.

It is made by placing hydrogen, sodium and phosphorus atoms with an odd number of nuclear particles in a magnetic field. These nuclei behave like small magnets and align with each other in the field. Upon receiving a stimulus of a radio energy pulse of a specific frequency, determined by the intensity of the main magnetic field, the nuclei are reoriented with respect to the axis, emitting radio frequency signals that generate the image and are transformed with a computer, where an intense contrast is observed between the different tissues. MRI images can be obtained in the axial, coronal, sagittal and oblique planes.

MRI provides numerous advantages over CT; Firstly, the possibility of obtaining images directly, and therefore with the highest quality, in any plane of space, both of the brain and the spinal cord. MRI has a much greater resolution capacity than CT, which allows the detection of very small lesions, as well as those of the white matter (demyelination). Compact bone does not produce a signal on MRI, so there are no artifacts that prevent the basal structures of the brain (pituitary, pituitary stem, brain stem and cranial nerves) from being seen with extraordinary clarity. Perhaps the area where the contribution of MRI has been most decisive is in diseases of the spine and spinal cord, places in which it allows images of definitive diagnostic value, without contrast and without risk for the patient.

For all these reasons, MRI is progressively displacing CT for the diagnosis of most diseases of the brain (especially in certain tumors and malformations of the posterior fossa, conditions of the sella turcica and cranial nerves, hydrocephalus and demyelinating diseases) and of course, of the spinal cord.

(alterations of the cervicocranial junction, spondylotic myelopathy, syringomyelia, spinal tumors and dorsal disc herniations). CT information is usually sufficient in disc pathology of the lumbar region.

MRI is more expensive than CT. The examination takes longer and patients who suffer from claustrophobia do not collaborate in the examination. It cannot be performed on people with pacemakers, large metal prostheses or vascular surgical clips that are ferromagnetic.

EXPLORATION OF THE CENTRAL NERVOUS SYSTEM USING RADIOISOTOPES

BRAIN SCAM

It was introduced by Moore in 1948. It consists of using substances such as diethylenetyraminopentacetic acid (DTPA) labeled with radioactive material such as In.13 This radiopharmaceutical is injected intravenously and after 10 minutes images can begin to be obtained with equipment called a gamma topographer. Any views that are necessary are performed according to the clinical data.

It is used in the diagnosis of primitive or metastatic brain tumors. It is more effective in supratentorial tumors. It has also been used in the diagnosis of cerebral vascular accidents.

It has the advantage of being a bloodless investigation that is easy to carry out. It offers a high percentage of positivity, but a negative test does not negate the existence of a brain tumor, especially if a posterior fossa tumor is suspected.

The process of accumulation of the radiopharmaceutical in the tumor tissue is considered related to the vascularization of the tumor, cellular metabolism and other factors.

beef.

POSITRON EMISSION TOMOGRAPHY (PET)

Although the clinical use of positron emission tomography (PET) is expanding, it is currently only generally used as a research technique in specialized centers in some European countries, the United States, and Japan. The difficulty of its clinical use and its high cost are due to the need to use isotopes with very short half-lives, which requires each equipment to have a unit (cyclotron) capable of continuously synthesizing them as needed; This represents a very high cost in equipment and personnel for its operation and maintenance. Its poor capacity for anatomical resolution is another drawback of current PET equipment.

In any case, this exam offers very useful information that is impossible to obtain any other way. By using oxygen and carbon isotopes, cerebral blood flow and local oxygen extraction can be studied regionally; This data, together with the local consumption of glucose labeled with Fl8, allows the study of neuronal metabolism. With these techniques, hypometabolic areas are detected in the foci of ischemia or in the epileptic foci in an interictal state, not detectable with imaging techniques (CT, MRI), which may be normal. Something similar occurs in dementias in which the hypometabolic areas correlate with the clinical symptoms and the neuropathological bases of the process. For example, they affect the frontal region in Pick's disease, frontal type dementia and Huntington's chorea, while the hypometabolic areas in Alzheimer's type dementia are bitemporoparietal.

Another possibility offered by PET is to label some neurotransmitters, their precursors or agonist-antagonists with isotopes. The most proven have been those relating to dopaminergic metabolism, which has made it possible to demonstrate the loss of L-dopa uptake by the striatum in Parkinson's disease and other rigid-akinetic syndromes, as well as the state of striatal dopaminergic receptors. The status of other receptors, muscarinic, GABAergic, etc., is beginning to be studied in other processes.

PHOTON EMISSION TOMOGRAPHY (SPECT)

SPECT is a tomographic technique that is based on the detection of photons emitted by a radioisotope, generally [23, It uses a gamma camera with one or more detector heads. It is much cheaper and more accessible than PET. The most commonly used ligand is an amphetamine derivative, which is very fat-soluble and crosses the blood-brain barrier, allowing the radiotracer to be distributed proportionally to cerebral blood flow; For this reason, it can detect areas of decreased flow like PET, although it cannot determine the degree of metabolism. SPECT can also detect areas of tracer accumulation due to disruption of the blood-brain barrier in the case of arteriovenous fistulas, flow perfusion or collateral circulation in acute infarctions, primary tumors or metastases, and other conditions.

nes.

ELECTROENCEPHALOGRAPHY

This exploration was put into practice in 1929 by H. Berge, when introducing the electroencephalogram (EEG), which, despite its limitations, has become a very valuable resource in the study of patients neurological, as long as the results are not considered isolated, but integrated with the clinical data.

The EEG consists of graphically recording brain electrical activity using electrodes placed on the scalp. As can be understood, it is a study that does not imply any risk for the patient and can be performed in a short time and at any age.

It is mainly used to obtain evidence of focal or diffuse disorders of brain electrical activity, produced by structural lesions or biochemical alterations. As psychiatric syndromes of emotional origin (which are not caused by lesions of the central nervous system) are not accompanied by electroencephalographic alterations, the EEG is also used to distinguish these psychiatric disorders (called functional) from psychiatric disorders produced by organic brain lesions.

The EEG is a fundamental investigation for the study of epilepsy and seizure syndromes.

Neurons, whose approximate number is two and a half billion, are in constant electrical activity generating action potentials that in turn guarantee the multiple interconnections between this immense number of cells.

Although the basic mechanisms that result in electroencephalographic recordings are not well known yet, it is accepted that they are produced by the integrated or resulting value of membrane potentials, cell body potentials, axonal and dendritic potentials and, probably, neuroglial potentials. It is as if we were listening to the music performed by thousands of orchestras at the same time; This resulting noise is the electroencephalographic tracing. This is a limitation, to which is added the fact that it is recorded on the scalp and not directly on the brain, and that electrodes are placed only at certain points. Although their position is aimed at collecting cerebral cortical activity, we have seen that there are other systems (ascending activating reticular system and diffuse thalamic projection system) that exert a notable influence on the cerebral cortex.

The EEG is characterized by a continuous and constant change of potentials in different areas. If electrodes are placed in two different places on the skull, they will pick up the relative potential differences between those areas. These potential differences, collected by electrodes located in conventionally agreed locations on the skull, are passed to an amplification system and from this to fine, inscribing needles, whose movements are recorded on a special paper, which passes under these needles at a uniform speed (1.5 cm/s).

The electrical activity collected by the electrodes is of the order of 50 uV (microvolt), so it has to be amplified up to a million times.

NORMAL ELECTROBRAIN GRAM

It varies with age, with the state of consciousness (sleep or wakefulness) and presents different characteristics in frequency and amplitude in the different areas of the skull where it is collected.

The main normal rhythms and waves are: alpha and beta.

Alpha rhythm. 8-13 cycles/s. It predominates in occipital regions, with the patient awake and eyes closed.

Beta rhythm. More than 13 cycles/s (14-30); It replaces the alpha rhythm when the patient opens his eyes, thinks actively or is in a state of attention to external stimuli.

The amplitude of its waves is smaller than that of the alpha rhythm, and its speed is greater. It predominates in the anterior regions of the head. Like the alpha rhythm, it tends to be symmetrical and equal on both sides of the head.

The sleep EEG is characterized by slow waves, the so-called sleep spindles, generalized slow activity or faster activity, depending on the depth and periods of sleep.

The electroencephalogram in the newborn is characterized by low voltage slow waves. Around the age of 5, the alpha rhythm appears, which progressively extends; and towards puberty the beta rhythm is established regularly.

ABNORMAL ELECTROENCEPHALOGRAM

Delta rhythm. 0.5-3 cycles/s; It appears as a complex of irregular, persistent waves that are not modified by opening the eyes or hyperventilation.

They come from a group of abnormal cortical neurons, which discharge their electrical activity irregularly.

They are produced by expansive masses (neoplasms, hematomas, abscesses) or trauma. In these cases the slow waves are unilateral.

Theta rhythm. 4-8 cycles/s; It may appear during sleep or fatigue. It is also observed in some forms of epileptic discharges, drowsiness, anemia, cerebral anoxia, cerebral atrophy and in senile people. In these cases the slow activity is bilateral.

Spikes or points. It is one of the best-known pathological waves. This is a rapid discharge (20-60 ms) of higher voltage, which stands out as a larger, narrower wave that ends in a pointed shape.

It is the typical wave of grand mal epilepticus, in which case multiple spikes appear, one after the other, which replace the normal rhythms in all the areas where the brain's electrical activity is collected. The spikes can be located, then indicating their cortical origin. They are common in so-called focal or partial epilepsies, which originate in the cerebral cortex. In these cases they usually be produced by the various types of injuries that can cause epileptic seizures.

Other discharges originating in deeper structures have a less sharp shape than the spike when they are collected at the surface (sharp waves). Sometimes spikes are combined with these sharp waves, or with slower waves.

The combined wave-spike or spike-wave rhythm of 3 cycles/s is characteristic of petit mal epilepticus (typical absence).

INDICATIONS

Epileptic seizures. The electroencephalogram, when positive in the presence of clinical seizures, confirms the diagnosis and is useful for classifying, together with the clinical data, the type of epilepsy that the patient presents, and for monitoring the patient' s evolution. One or more normal electroencephalograms do not deny the existence of epilepsy. In cases of focal epilepsy, the electroencephalogram cannot specify the nature of the lesion that produces the epileptic discharge.

Intracranial expansive mass. It produces electroencephalographic alterations that allow us to suspect the existence of a tumor mass in a certain location (slow waves in a fixed focus), but the electrical alterations will be similar in neoplasms, abscesses or hematomas, assuming that their size and growth are similar.

Other disorders. The electroencephalogram can be pathological in cranial trauma, meningoencephalitis, intracranial hypertension, cerebral atrophies, hepatic coma, cerebral anoxia and other conditions.

Determination of brain death. Here a flat electroencephalogram is

Finally, we must take into account that an electroencephalogram may be normal and the patient may have an organic brain pathology; Conversely, normal people can be carriers of a pathological electroencephalogram. These circumstances, together with the fact that a pathological electroencephalogram only allows us to affirm that there is a brain dysfunction, without which we can diagnose specific pathologies of an injury nature, mean that (as we said at the beginning) the electroencephalographic results must always be evaluated in light of the clinical data. Under these conditions, it is a very useful test in medical practice.

EVOKED POTENTIAL

They are the electroencephalographic responses to different types of intense sensory stimuli that produce the simultaneous activation of numerous receptors.

The most common evoked potentials in clinical practice are:

1. Visual evoked potentials (VEP). These potentials are evoked by stimulating the retina with repeated inverse checkerboard patterns.

   PEVs are recorded separately in each eye.
2. Brainstem auditory evoked potentials (EBAT). They are commonly evoked by brief clicking sounds in one ear, while a random noise is emitted in the other. The normal response recorded by an electrode in a vertex reference ear consists of a series of submicrovolt waves, approximately at 1 mm intervals.

3. Somatosensory evoked potentials (SEP). They are evoked by brief stimuli in any of several sensory nerves and dermatomes; although generally the median, peroneal and posterior tibial nerves are used.

Due to their small amplitude, the evoked potentials cannot be directly observed in an electroencephalographic recording, but rather the responses to various stimuli need to be averaged, using special equipment, so that these potentials can be clearly defined.

INDICATIONS

Suspected intracranial tumor (pituitary tumors, cerebellopontine angle tumors, etc.), neurodegenerative diseases (oligopontocerebellar atrophy), epilepsies, head trauma, multiple sclerosis, metabolic disorders (vitamin B'2 deficiency), delays in the development of the newborn and diagnosis of brain death.

INFORMATION YOU CAN PROVIDE

1. Detect failures in the anatomical or functional integrity of the sensory system being explored.
2. Its quantitative study allows the presence of lesions to be identified more accurately than the visual report of the electroencephalogram.

ELECTRONMYOGRAPHY AND ELECTRICAL STIMULATION OF PERIPHERAL MUSCLES AND NERVES

It consists of recording the electrical potentials produced by the muscles in rest and activity using electrodes inserted in them, the electrical activity passes to a cathode ray oscilloscope and a sound reproducing equipment, for simultaneous vision and hearing of the muscular activity:

In normal muscle at rest, no activity is detected, but during voluntary contraction the action potentials of the motor units are obtained.

A motor unit is understood to be the union of the axons of the motor neuron (second neuron) and the muscle fibers that innerva; It is composed of the following parts: neuronal body, axon, neuronal junction or neuromuscular plate (myoneural) and muscle fibers.

Electromyography is pathological in diseases that affect this motor unit, with alterations appearing both at rest and in muscle activity. If it is combined with electrical stimulation of peripheral nerves and muscles, it is sometimes possible to locate the process in the different parts of the motor unit and suspect its cause. However, it should be clarified that using only the data from the electrical study, a diagnosis of a nosological entity cannot be made. There are no pathognomonic waves of diseases. Electromyography, like the electroencephalogram, helps in the diagnosis, but its results must be integrated with the clinical history and the results of other tests, which will allow conclusions to be reached.

INDICATIONS

Muscle diseases (primary myopathies). Sometimes there is difficulty in clinically differentiating muscle atrophy caused by primary muscle disease and amyotrophy due to denervation (neuropathic atrophy). Electromyographic results are typical. There is no electromyographic difference between muscular dystrophies and myositis.

Neuromuscular transmission disorders. The electromyogram is of value in the diagnosis of myasthenia gravis, since it can show evidence of disorders in the myoneural plate. Although similar electromyographic disorders can be seen in neuronal diseases such as amyotrophic lateral sclerosis, these show other alterations that myoneural plate conditions do not present.

Diseases of peripheral motor neurons. Even before there is clinical evidence of these diseases, electromyographic data of denervation can be obtained. This examination allows the muscle groups to be explored separately, so the extent of the process can be known with precision. Electromyography can provide information that helps determine whether the condition is a root, a plexus, or a nerve of several nerves; and to differentiate these alterations from those that affect the neuronal bodies in the anterior horn of the cord.

Simulated paralysis or paralysis that appears in a conversion hysteria. In cases where the clinic alone does not allow such conditions to be assured or the doctor is required from a legal point of view, normal electromyography can be definitive, by specifying the nature of both.

BIOPSIES

NEUROMUSCULAR BIOPSY

Nerve and muscle biopsy are frequently combined and are a decisive means in the diagnosis of diseases of the peripheral nervous system and muscle.

Several sensory nerves are accessible to the nerve biopsy with few consequences for the patient: if the biopsy is fascicular (the entire nerve is not cut), the residual sensory deficit is minimal. Despite this, the patient should be warned of this risk and the possibility of a painful neuroma remaining. Generally, the biopsy is performed on the sural nerve, behind the external malleolus of the ankle. It can be done in the middle third of the leg and, with the same incision, take a biopsy of the peroneus brevis muscle. This technique has the advantage that only one wound is made, but it has the disadvantage that the nerve at that level can be very traumatized, because it is very superficial. Precisely, the advantage of taking it from behind the ankle is that it is very protected from external trauma and that, in addition, good normalized histograms are available on the distribution of different populations of fibers in that nerve.

The biopsy must be done very carefully so as not to produce artifacts due to crushing or stretching of the fibers. The excised nerve segment should be processed for light, electron, separated fiber microscopy, and immunohistochemistry. In addition to the descriptive study, histograms of the fibers must be made; The diameter, density and number of myelinated fibers can be studied in the semi-thin sections and the unmyelinated fibers in photomicrographs of the ultra-thin ones; The length of the segments and its relationship with the diameter of the fibers is studied in the separated fibers. These techniques are expensive and time-consuming, so fully exploiting a nerve biopsy is only within the reach of well-equipped laboratories.

The findings are nonspecific in many axonal polyneuropathies that can be diagnosed by the clinical picture, the electroneurogram-electromyogram and the laboratory; for example, diabetic, uremic, alcoholic, porphyric, drug or toxic polyneuropathies;
In all these cases, nerve biopsy is not indicated except in specialized settings with research programs. Even in many familial polyneuropathies, clinical and neurophysiological diagnosis is sufficient for daily practice. Nerve biopsy is very useful in subacute polyneuropathies, with clinical suspicion of inflammatory demyelinating polyradiculoneuritis or paraproteinemia. Histological images are diagnostic in panarteritis nodosa, leprosy, amyloidosis and lymphomatous or neoplastic infiltrations.

54

MAIN SYNDROMES OF THE SYSTEM

HIGHLY STRUNG

The most frequently observed neurological syndromes in the clinic

Are:

- Motor syndromes.
- Sensitive syndromes.
- Brain stem syndromes.
- Cerebellar syndrome.
- Meningeal syndrome.
- Endocranial hypertension syndrome.
- Comatose syndrome.

MOTOR SYNDROMES

Among the motor syndromes we have:

- Hemiplegic syndrome (studied in “Motility disorders”, Chapter 51).
- Paraplegic syndrome.
- Extrapyramidal syndrome (involuntary movements).
- Parkinsonian syndrome.
- Convulsive syndrome.

PARAPLEGIC SYNDROME

Concept

Paraplegia (crural or inferior paraplegia) is called paralysis of both legs, due to neurological causes.

In a considerable percentage of patients, the paralysis of the legs is not total, and in these cases it is called paraparesis.

Paraplegia can be established in two ways: suddenly or slowly; The interrogation must specify this mode of beginning, which as we will see is important to diagnose its location and cause.

Clinically, there are two main forms of paraplegia: flaccid and spastic, which have different symptoms, production mechanisms and causes, so we will study them separately.

The general characteristics of the two main forms of paraplegia are shown comparatively in Table 54.1. In both forms, sphincter disorders, objective sensitivity disorders, or both may or may not exist.

Table 54.1 General characteristics of the two main forms of paraplegia

Parapl e íafl actta	Spastic parapl gj ía
1. Loss of muscle strength	1. Loss of muscle strength
2. Hypotonia and muscle atrophy	2. Hypertonia (muscle spasticity)
3. Hyporeflexia or arrhythmia osteotendinous flexia	3. Osteotendinous hyperreflexia
4. Not Babinski	4. Babinski
5. Don't clone	5. Clone

In addition, a third type of paraplegia is described, the so-called flascospastic paraplegia, which does not present muscle hypertonia, but in which there is a Babinski sign and sometimes hyperreflexia. In reality, it is a spastic paraplegia that initially presents (because of its abrupt onset) a stage of muscle flaccidity.

flaccid paraplegia

Syndromogenesis or pathophysiology

Flaccid paraplegia is caused by an injury to the second motor neuron, the anterior horn neuron of the spinal cord or spinal motor neuron. Their functions are then abolished, among which two important ones, muscle trophism and the spinal reflex arc, are quickly affected (amyotrophies and areflexia). If the injury to this neuron occurs directly in the anterior horn where the cell bodies are, or in the anterior roots, the disorder will be purely motor (example: poliomyelitis). If the lesion occurs in its axon, after the anterior root, there will be associated sensory disorders, since the axons are part of the peripheral nerves that are mixed (polyneuropathy).

In these cases there are no sphincter disorders, since the spinal centers and pathways that govern the sphincters are not affected. A special case is the flaccid paraplegia that can accompany the cauda equina syndrome, in which sphincter disorders are marked (see 'Alterations of sensitivity', Chapter 51) .

Muscle tone, the result of pyramidal, extrapyramidal and cerebellar influences, is affected (hypotonia) because all these functions are carried out through this second motor neuron. There is no Babinski because the pyramidal pathway is intact.

Syndromography or positive diagnosis

Clinical picture

- Functional impotence and loss or decrease in muscle strength. Sometimes walking is impossible, but when it is achieved, it can be steppe or almost normal, especially if it is a paraparesis.

There is muscle hypotonia, the muscles are soft and passive movements are performed with less resistance.

There may be amyotrophy, variable depending on the cause and time of evolution.

There is no areflexia or hyporeflexia.

- There is no Babinski sign.

Additional investigations

Electromyography shows signs of degeneration.

Etiology

1. Polyneuritis (various causes).
2. Guillain-Barré polyneuroradiculitis.
3. Acute anterior poliomyelitis or Heine-Medin disease (non-existent condition in Cuba).
4. Charcot-Marie-Tooth disease (progressive peroneal muscle atrophy).

Spastic paraplegia

Syndromogenesis or pathophysiology

Spastic paraplegia is caused by injury to the pyramidal pathway at the level of the spinal cord; therefore, the first motor neuron is injured.

As in any injury to this neuron, there will be hypertonia, hyperreflexia and clonus. As is known, the Babinski sign is pathognomonic of pyramidal lesions. Accordingly, flask-pastic paraplegia is also a paraplegia due to injury to the first motor neuron, in which there is a functional inhibition of the second motor neuron, related to the sudden form of spinal cord compression or injury. (This paraplegia appears in other texts as spinal flaccid paraplegia.)

Therefore, the types of paraplegia resulting from spinal cord injury or compression are directly related to the intensity of the spinal injury and the speed with which this injury has occurred.

There are paraplegias with loss of motility, sensitivity and reflexes below the injury, which although functionally this corresponds to a spinal cord section, anatomically the injury does not exist, which leaves open the possibility of recovery (spinal cord shock).

In cases of slow spinal compression, as this generally occurs from outside the spinal cord, the spinal roots are compressed first, which explains the onset of radicular pain.

In spastic paraplegia due to myelopathy, the lesion may be limited to the pyramidal tract in the spinal cord, for which reason sensory and sphincter disorders may be absent.

Syndromography or positive diagnosis

Clinical picture

It is identified, as we said before, by: motor paralysis, contracture (hypertonia), hyperreflexia, clonus and Babinski sign. Trophic disorders are rare, except if the condition is long-standing.

Spastic paraplegia can take two clinical types: extension and flexion.

Spastic extension paraplegia is the most common. Paralysis is generally less intense than in flaccid paralysis, walking is difficult or impossible (digitigrade gait). There is clonus and marked hyperreflexia. There is a Babinski sign. There may or may not be spinal automatic reflexes (withdrawal of the lower limb in triple flexion in response to a painful stimulus), but they are generally absent. There are sphincter disorders. Sensitivity disorders may be absent or present.

Spastic paraplegia in flexion is commonly the evolutionary result of spastic paraplegia in extension. A contracture is observed in triple flexion of the legs, impossible walking, reflexes abolished due to flexion. There are Babinski and sphincter disorders, as well as very marked spinal automatic reflexes.

These two types of paraplegia correspond to slowly evolving spinal cord compression and, less frequently, to an intrinsic injury to the spinal cord.

Compression paraplegia is characterized by:

1. Signs of root compression:
    a) Fixed sitting pain, unilateral or bilateral, that reaches great intensity. Against a background of dull pain, very painful paroxysms are interspersed that appear with coughing and sneezing.
    b) On examination there is hyperesthesia; Later, band hypoesthesia occurs, which demonstrates its root topography. These signs belong to the paraplegic state.
2. Spinal automatism reflexes.
3. Beginning with Brown-Sequard syndrome.
4. Sensory disorders that allow the spinal level of compression to be specified.
5. Constant sphincter disorders.

Paraplegia due to intrinsic spinal cord injury is characterized by the absence of signs of root compression and spinal cord automatism. Sensory and sphincter disorders may be absent.

Etiology

1. Spastic paraplegia due to slow spinal cord compression:
    a) Vertebral cancer.
    b) Spinal tumors.
    c) Pott's disease.
2. Spastic paraplegia due to myelopathy (intrinsic spinal cord injury):
    a) Multiple sclerosis.
    b) Pernicious anemia.
    c) Spasmodic spinal paraplegia.
    d) Syringomyelia: paraplegia is accompanied by a syringomyelic dissociation of sensitivity.

Flascospastic paraplegia

If the spinal compression is sudden, a phlascospastic paraplegia will occur.

Indeed, the most typical form of this paraplegia is the so-called complete spinal cord transection syndrome or Bastián syndrome, which most often recognizes a traumatic etiology.

The paraplegia in these cases is total; All movement is impossible below the section and there is total anesthesia and flaccidity.

There is areflexia, Babinski sign and marked sphincter disorders. Trophic disorders are intense, they are accompanied by so-called pressure ulcers.

cough or bedsores.

Etiology

1. Complete spinal section.
2. Traumatic spinal shock.
3. Non-traumatic acute or subacute spinal cord compression.
4. Acute transverse myelitis.

EXTRAPYRAMIDAL SYNDROME (INVOLUNTARY MOVEMENTS)

Anatomophysiological count

The extrapyramidal system is responsible for the regulation, integration and control of involuntary motor activity. This system should be considered as an integrative system that contributes to the mechanism of movement, muscle tone and automatism, and not as an independent system.

It is composed of a set of complex suprasegmental structures, closely related to each other by abundant connections that form an intricate integration system between the cerebral cortex, the cerebellar cortex and the spinal cord.

It differs from the pyramidal system in that it has short routes, which make stops; However, they have a common endpoint, which is the motor neuron of the anterior horn of the spinal cord.

The following links intervene in its constitution:

1. Cortical link. Consisting of the somatosensory, motor and premotor areas and the cortical areas of posterior parietal, temporal and cortical association, from where it originates and projects to the striopalidal system.

2. Nuclear link. It is made up of six pairs of basal nuclei: the caudate nucleus and putamen (striatum), the globus pallidus, the subthalamic nucleus (body of Luys), the red nucleus and the substantia nigra.
   The nuclei of the mesencephalic roof, the inferior olivary nucleus and the reticular substance also participate in its formation.

3. Internuclear connections. The internuclear connections are:
   a) Impulses from the thalanmus coming from the cerebellum and the reticular formation arrive to the striatal system (caudate nucleus and putamen).
   b) The caudate nucleus sends fibers to the putamen and the globus pallidus.
   c) Fibers destined for the pallidum originate from the putamen and send connections to the red nucleus, the subthalamic nucleus (Luys) and the substantia nigra, to the nuclei of the roof and to the inferior olivary.
4. The afferent pathways. They are explained at the bottom of fig 54.1.
   In summary, the extrapyramidal system receives its stimuli (afferents) from the following four sectors: cerebellum; reticular system, internal lemniscus (sensory pathway) and cerebral cortex. In reality, in the current state of knowledge, one cannot speak of a well-defined "path" but rather of links that time will clarify.

5. Discharge or efferent pathways. We must consider without much conceptual rigidity that a large part of the outputs are made through the internal pallidum (fig. 54.2); but they are not the only ones. I will consider-
   We use the following routes, whose routes go:
   a) From the internal pallidum to the ventrolateral nucleus of the optic thalamus, which in turn redirects the impulse to the precentral brain region.
   b) Towards Luys' body, which, for its part, receives impulses from the opposite side.
   c) Towards the red nucleus.
   d) Towards the posterior substantia nigra.
   e) Towards the reticular formation (of the midbrain, pons and medulla).
   f) To the hypothalamus.
   g) To Darkshevich's core, on the opposite side.
   h) To the nuclei of the motor nerves of the face, eyes and chewing nerves.

Accepted that these nuclei receive afferents, the discharge or efference is carried out at eight different levels: cerebral cortex (through the ventral anterior nucleus of the thalamus), body of Luys, red nucleus, posterior substantia nigra, reticular formation, hypothalamus, motor nuclei of the face and nucleus of Darkshevich.

From these various points, and through pathways that are not well clarified, the extrapyramidal system is incorporated or integrated into the pyramidal pathway, along the way or at the level of the corresponding final common pathway, either in the brainstem or in the anterior horns of the spinal cord.

The red nucleus constitutes within the pyramidal system an important link station between the cerebral cortex, the striatum, the cerebellar cortex and the motor neuron of the anterior horn of the spinal cord:

- The rubrospinal bundle originates in the red nucleus, almost immediately after its birth, decussates at the level of the cerebral peduncle, descends on the opposite side of the pons and the medulla oblongata, and then along the lateral cord of the spinal cord, to reach the motor neuron of the anterior horn of the cord.

The tectospinal tract begins in the nuclei of the roof, decussating and descending on the opposite side of the pons, medulla oblongata and lateral cord of the cord, to end in the anterior horn of the spinal cord.

The vestibulospinal tract arises in the vestibular nuclei (Dieters nucleus). It descends on the same side of the spinal cord and ends the same as the previous one.

The reticulospinal bundle arises in the cells of the reticular formation of the pons. There are two: the direct bundle, which descends on the same side of the cord, and the crossed bundle, which descends on the opposite side and ends in the anterior horn of the spinal cord.

Physiological studies have shown that one of the main roles of the basal nuclei is to gradually increase the amplitude and speed of movement and not initiate it although it participates in its preparation.
ration.

The disorder of the basal nuclei impairs movements, causing various types of abnormal movements.

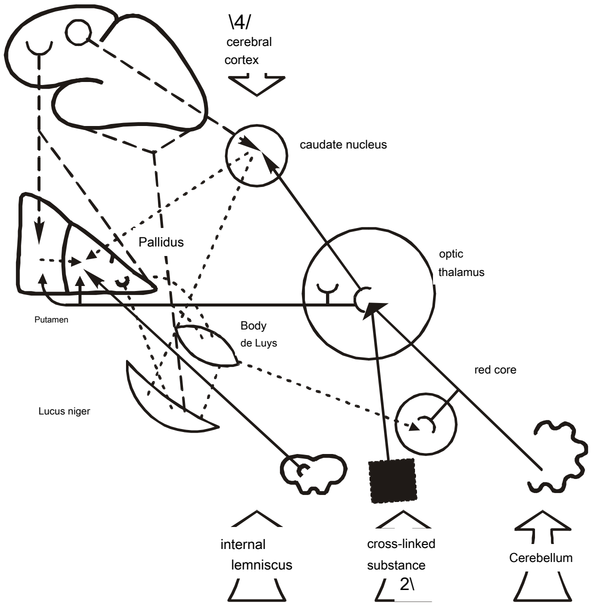
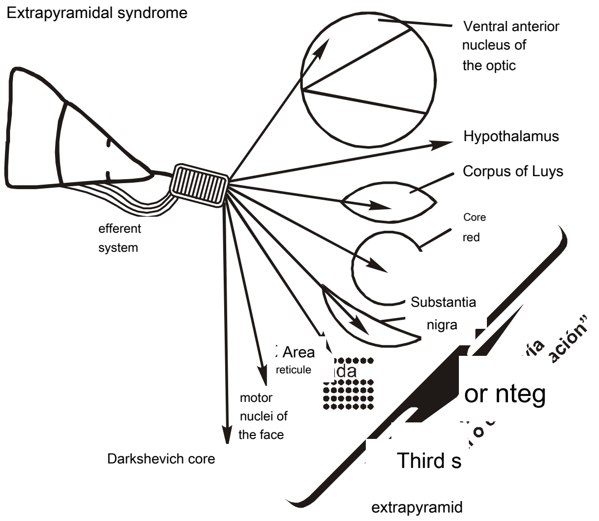

\4/
cerebral cortex
caudate nucleus
Pallidus
optic thalamus
Putamen
Body de Luys
red core
Lucus niger
internal lemniscus
cross-linked substance
2\
Cerebellum

Fig. 54.1 Scheme of the afferent pathways of the extrapyramidal system (a solid line corresponds to the true afferents): 1, comes from the cerebellum, but discharges or reinforces elements in the optic thalamus; 2, from the reticulated substance it passes, like the previous stimulus, through the optic thalamus;
3, brings contributions from the internal lemniscus (sensitive pathway); 4 (marked by lines and dots), constituted by the contribution of the cerebral cortex. On the dotted line we have marked the connections.

Fig. 54.2 Scheme of the efferent pathways of the extrapyramidal system. We send the largest contingent of efferents through the pallidus and putamen, although they also do so from other nuclear systems. In summary, the connections are made through eight paths, but whatever the path followed by these paths, all of them must finally be incorporated to integrate a third sector, before reaching their final destination. Its incorporation would be done in the various links of the motor activity pathway, tone, balance, coordination, etc. (This scheme is not intended for specialized doctors.)


Extrapyramidal syndrome
Ventral anterior nucleus of the optic
Hypothalamus
Corpus of Luys
Core red
efferent system
Substantia nigra
Area reticule
da
or nteg
motor nuclei of the face
Darkshevich core
Third s
extrapyramid

Extrapyramidal movement disorders are divided into:

- Hypokinesias; characterized by decreased and slow mobility.

- Hyperkinesias; It is manifested by abnormal involuntary motor movements.

Abnormal movements or hyperkinesias

Abnormal movements or hyperkinesias can present in different forms and characteristics. Some are easy to define, but not others that are produced by the combination of several of them.

1. These are involuntary movements, caused by muscle contractions.
2. In certain circumstances they allow, due to their semiological aspect, the location of the altered anatomofunctional system.
3. They can occur in sectors of the body or throughout the body.
4. They are sometimes the result of lesions of the cerebral cortex (motor and premotor area), its descending pathways, gray nuclei of the base, brainstem (peduncle, pons and medulla), cerebellum and its pathways, spinal cord, peripheral nerves or the muscles themselves.
5. They have an organic character (although there are also functional ones) and linked to various processes: infectious, toxic, degenerative, traumatic, tumorous or occurring in the course of genetic diseases.
6. The character of the movements generally depends on two circumstances:
   a) From the site of the injury, but we must remember, however, that injuries in different locations can cause the same type of movement.
   b) The type of injury, although it is also necessary to clarify that injuries in a certain sector of the neuroaxis can cause various types of abnormal movements.
7. Inspection generally allows semiological diagnosis of the type of movement. In this sense, it is necessary to observe the following details:
   a) What part of the organism is the seat of the abnormal movement, for example, the limbs (proximal or distal portion), the face, the neck, the back, etc.
   tera.
   b) What is the extension of the movement and what is its distribution (a single muscle or muscle groups), what joints are involved, etc.
   c) With respect to the rhythm, its uniformity or not, its regularity or not, whether it is continuous or intermittent, as well as its intensity, speed and amplitude.

d) Its connection with rest or movement, its relationship with mood changes, possible control or not of the will and disappearance during sleep (dream calm or acalmia, described for the first time by Marotta, an Argentine neurologist).

8. Many times, after describing the details stated in the previous paragraph, it is now possible to call the movement abnormal.
9. The inspection is completed with palpation of the various muscle groups, which may be found in hypotonia (this is the case of choreic movements), with increased muscle tone (for example, in spasmodic torticollis) or, on the contrary, with alternating muscle tone (hypertonia alternating with hypotonia), which is the so-called dystonia.

10. The study can be completed with cinematography or electromyography, resources that are outside what the general practitioner needs.

It is difficult to establish a classification of the various abnormal movements, since sometimes they are found in a state of purity and in other circumstances they are combined with each other. For this reason, we are going to limit ourselves to the description of the most frequent ones, which are:

- Tremors.
- Myoclonus.
- Choreic movements.
- Dystonokinesias.
- Muscle spasms.
- Tics.
- Hemiballism.
- Fasciculations and fibrillations.
- Seizures.
- Psychogenic hyperkinesias.

Tremors

They are characterized by a series of involuntary movements, of all or a part of the body, around its equilibrium position. It is the result of alternating and rhythmic contractions of muscles that have opposite functions.

1. Exploration. Sometimes the tremor is evident by simple inspection; However, in other cases certain maneuvers are necessary. The following are advisable:

a) The patient is invited to extend forward, horizontally, the two upper limbs in the classic attitude called "swearing", the fingers They will be separated. If the tremor does not appear in that position, it is advisable to place a piece of paper over the fingers. If there is tremor, it is possible that it is transmitted to the paper and thus becomes more noticeable.

b) Another way to detect the so-called intentional tremor (that is, the one that appears in the course of a voluntary act), consists of the patient executing certain movements (touching the tip of the nose or drinking a glass of water). In these circumstances, it is noted that the movement begins correctly and goes in the appropriate direction, but as the movement continues, a series of oscillations appear before reaching the indicated end.

c) The patient is asked to extend his upper limbs in the so-called "swearing position", while the doctor places the palms of his hands in contact with the tips of the patient's fingers.
It is sometimes possible to see the existence of small oscillations.

d) The examination can be completed by asking the patient to write down what is dictated to him, so that it can be seen if there is no tremor. The study of tremor must also include different steps to determine: its location (limbs, face, etc.); its extension (segmental, hemilateral or generalized) and its topography (distal, proximal or total); its width (in millimeters or centimeters); their rhythm (Charcot classified them as fast, 8-9 oscillations/s: medium, 5-7 oscillations/s: slow, 4-5 oscillations/s): their relationships with voluntary movements (appearance, increase, decrease, disappearance); These details are important (when oscillations appear in the course of voluntary movements, they are called "intentional tremor"); finally, its existence at rest.

2. Types. From a practical point of view, tremors are classified as follows:

a) Rest. As their name indicates, they occur when the patient does not carry out any activity.
The best example is that described in parkinsonian syndromes and Parkinson's disease:
the fingers are slightly flexed; the thumb in adduction, resting on the terminal phalanx of the index and middle fingers, giving the impression of making breadcrumb balls or rolling cigarettes.

Other times the fingers move little or not at all; On the other hand, those that tremble are the hands, which "beat the drum." In the lower limbs, tremors

They can occur on the feet, either in the form of clicking heels, or following the rhythm of an endless and monotonous beat, less frequently, relying on the tips of the fingers, which strike the ground. A tremor of the lower jaw may be observed (this is the maxillary phenomenon of the extrapyramidal) or an incessant movement of the tongue, which shakes like a plunger. When both movements are combined (tremor of the lower jaw and shaking of the tongue) we have the linguomaxillary phenomenon described by Castex, Battro and Camauer as an extrapyramidal manifestation. The tremor of the upper limbs decreases and sometimes stops when the parkinsonian quickly executes a voluntary movement. This detail is important for the diagnosis. This type of rest tremor is found, as we already said, in Parkinson's disease and in parkinsonian syndromes (infectious, toxic, etc.).

As characteristics, we repeat, tremors of this type occur at rest, are monotonous and stereotyped. However, they do not appear in some parkinsonian symptoms when the contracture is such that it " suffocates" them.

b) Intentional or action tremors. They are easy to recognize, since during rest the patient does not present any abnormal movement, but it appears when he is instructed to carry out a certain segmental movement; for example, putting the tip of your finger to your nose (index-nose test) or having you drink water from a glass placed on the table, or taking a pencil and writing.

In all these movements we observe the following: the movement begins correctly;

The direction is good, but before reaching its objective, problems begin to appear. Sometimes it stops and resumes its path, or oscillations appear that end in tremors. At absolute rest, the segments of the examined limb are calm, so to speak. This type of so-called intentional tremor is found in numerous processes: plaque sclerosis, cerebellar diseases, Friedreich's disease (familial hereditary degenerative disease), certain focal lesions such as Benedikt's peduncular syndrome (due to damage to the red nucleus), Wilson's disease (hepatolenticular degeneration), etc.

tera.

c) Permanent tremor. It is observed during rest or is somewhat attenuated by maintaining a position (extended arms, for example) and persists without major modifications during execution.

tion of a voluntary or automatic act. Sometimes its value does not have, in certain circumstances.

great significance. Thus, we find the physiological tremors of fatigue, cold, emotion. On the other hand, it is important in Basedow's disease where, although it is permanent, it is exaggerated with movements. It is also found in progressive general paralysis, chronic alcoholism, smoking, mercurial or coffee or tea poisoning. There is a hereditary familial tremor that is only progressive when a senile state is grafted onto it. It is possible to find it in elderly subjects.

Myoclonus

Myoclonus are sudden clonic contractions, limited to a muscle fascicle; more rarely, they concern the entire muscle, especially when it is small in volume. It is a contraction that is seen and felt, even if it is partial (some patients inform us from moment to moment, where they appear and when they disappear). In general, when they are partial, they do not tend to displace the sector where they are produced. However, on other occasions they can do so.

Thus, when they occur in the toes, they are flexed and extended. As an important characteristic, we emphasize that they are essentially individual, autonomous and do not simultaneously interest several muscle groups. They can occur at the same moment in the biceps, back muscles, pectorals, trapezius, etc. One fascicle contracts independently of the others. Various classifications of myoclonus have been attempted. In this sense, Van Bogaert, Radermecker and Titeca divide them into two groups, according to the level of histopathological alteration.

1. In a first group they place clonisms (or myoclonias of the Anglo-Saxons), which often involve several muscles, acting synergistically, frequently in a symmetrical manner (the bursts of jerks due to their repetition originate a great Jacksonian episode or access). The lesions would be subcortical of the forebrain, from the midline to the interstriate defile.

2. A second group would be constituted by true myoclonus (in the sense of Friedreich and Unverricht) that frequently affects muscles or muscle fragments, without synchronism or symmetry, without rhythm and sometimes in bursts, and which, due to their repetition, give rise to painful contractures.
   Its anatomical location would be at the level of the olivodentate sector and, to a lesser extent, at the nigric and

thalamic. For the aforementioned authors, the two types described can become intricate.

Myoclonus can be found in encephalitis, especially in encephalitis of the algomyoclonic variety of Sicard and Roger. They can be seen in localized encephalitis and some of the abdomino-diaphragmatic type are observed (causing hiccups) or at the level of the tongue, or the eyeballs. Tuberculous meningitis at its onset may present myoclonus.

choreic movements

These are movements that respond to the following characteristics: they are involuntary, useless, unstoppable, uncoordinated and, furthermore, fast, brief, explosive. They occur during rest and increase in voluntary activities and emotion, disappearing during sleep.

They can cover a limb, half the body or the entire body, in addition to various sectors such as the trunk, face, tongue, lips, pharynx and larynx. The patient is unable to keep the tongue inside the oral cavity for any period of time; Speech loses its natural vocalization, creating noises rather than voices. There may be aphonia and also difficulty in chewing and swallowing.

An important fact for diagnosis is that all these movements can be combined. It should also be noted that during the physical examination, muscle hypotonia is an element that is never missing.

This type of movement is found in the following processes: acute chorea of the Sydenham type (or “Saint Vitus' disease”), acute infectious choreas secondary to eruptive fevers and well-defined infectious diseases, such as epidemic encephalitis. Chorea 0, or rather, choreic movements, instead of being generalized as in the conditions described above, can be unilateral, and in this form we find them in the following cases: superior syndrome of the red nucleus of Foix and Benedikt and Von Monakow syndromes (middle and inferior peduncular) .

We must leave a special section for Korea Cro.

Huntington's disease (a condition that occurs in adults, on whom a family burden weighs, which is progressive and ends in dementia). It is the antithesis of acute Sydenham's chorea, since it appears at school age, has no family incidence, has links with Bouillaud's disease, and if no complications occur, it cures within a period that can vary from two to three months.

As for the so-called chorea gravidarum, which, as its name suggests, occurs during pregnancy, For some it would be a Sydenham type chorea experienced in childhood that returns during pregnancy.

Dystonokinesias

These are movements that appear on the occasion of a voluntary movement or during the maintenance of an attitude; They are involuntary, they imprint sectors of the body or the entire body with new attitudes that will be maintained for a variable time (seconds or minutes) to decrease later until apparently everything comes into order, although a new abnormal movement is produced, and so on continuously. Since these movements appear when making a voluntary movement or adopting an attitude, they are called attitude dystonias.

For some authors, abnormal movements would be responses to paroxysmal increases in muscle tone at the level of variable muscle groups, without systematization and without rhythm. Below we explain the two clinical symptoms of dystonokinesias.

1. Spasmodic torticollis. The head is brought slowly, in jerks, to a vice position. Not by a single shake, but in several steps. Then the hypertonia appears to cease abruptly and the head tends to return to its normal position, although a new increase in muscle tone quickly takes it back to the abnormal position. These two stages of the process - bringing the head to an abnormal position and its return to normal - are repeated continuously; hence the name some gave it: rhythmic spasmodic torticollis. During the course of these movements, the well-known "antagonistic gesture" appears, more symbolic than effective; at first the patient opposes the twisting of his neck, holding his chin tightly in the opposite direction to the pathological movement, but the head is slowly carried as if a cabin boy were pulling invisible ropes. The patient then loosens his chin, temporarily abandoning it, although he maintains the habit of touching his chin on each trip.

   It will be remembered that we are describing spasmodic torticollis, which is a perpetual movement (except during sleep in which the patient adopts certain positions until before going to sleep, such as, for example, leaning on the bed with the sector of the face that is going to rotate, to immobilize it), as opposed to fixed torticollis, where the vicious position is permanent. Both have completely different etiological factors, pathogenic mechanisms and therapeutic behaviors.
2. Extended or generalized dystonokinesias. These are patients in whom the abnormal movements go beyond a simple spasmnodic torticollis, since, along with them, simultaneously,

abnormal movements in the tongue, muscles of the face, shoulders, limbs, or trunk. In general, they are due to lesions of the gray nuclei at the base. This group includes the following table:
dystonia or torsion spasm of the Ziehen-Oppenheim type (also called progressive lordotic dysbasia).

Someone has said that the sight of a patient of this type is unforgettable. According to André-Thomas and Ajuriaguerra, the phenomena present themselves in two forms: intermittent torsion spasm, which imparts rotational movements to the body axis and which we compare to that of a screw when it "enters a piece of wood", accompanied by attitudes of inclination, anteflexion and retroflexion, and permanent dystonia, which occurs when the patient attempts to adopt a bipedal attitude, walking, sitting or resting in dorsal recumbency.

muscle spasms

They are defined as a series of contractions of a muscle or group of muscles; often, almost all those innervated by a certain nerve. The spasm is rapid in onset and brief in duration, and may resemble muscle contraction induced by an electrical stimulus. In tonic spasm the contraction is longer or continuous, it may cause an altered position or be limited to a small movement. The spasm that limits movement can be defensive or protective. A painful, tonic, spasmodic contraction is usually called a cramp, which can cause defensive rigidity of the muscle. Spasms are usually of reflex origin, as a result of irritation produced at any level, from the cortex to the muscle. In many cases they are of peripheral origin, either at the nerve or muscle level.

Next let's look at some types of spasms.

1. Facial spasm. It can be, and is most frequently, secondary to a peripheral type facial paralysis that did not heal, due to lack of regeneration of the nerve fibers, on the one hand, and on the other, to an "error" in the direction of the cylinder axes of the nerve that were going towards regeneration. We have talked about peripheral facial paralysis and we have also said that facial spasm can occur as a consequence of it, but in other circumstances the presumed facial spasm will have to be interpreted as a form of Jacksonian seizure.
2. Hiccups. It is a brief spasm or reflex contraction of the diaphragm associated with the adduction or approximation of the vocal cords and accompanied by a strong inspiration. There is a sudden stoppage of breathing due to closure of the glottis, which produces a peculiar respiratory sound. It can be caused by irritation in a large area linked to the pneumogastric and phrenic nerves. Thus we find hiccups (due to irritations of the terminal part of the esophagus), gastric distention, pleuropulmonary, mediastinal or intrathoracic irritations, aortic aneurysm and also, abdominal irritation and distention. It can also be caused by alterations in the internal environment (acetonemia, uremia) or be a manifestation of encephalitis. Sometimes its origin is psychogenic.

Tics

Tics can be defined as a series of coordinated movements, which are repeated quickly, which mimic a "habitual voluntary act" and which involve a group of muscles in their normal synergistic relationships.
It is considered a habit, a spasm with a psychogenic background. Some tics are considered conditioned reflexes, originally precipitated by physical or emotional stimuli, reflexes that are then perpetuated as involuntary, stereotyped and recurrent movements. These are functionally grouped muscle contractions, accompanied by displacements or movements, which appear suddenly and are repeated at irregular intervals. Important fact:

They can, up to a certain limit, be controlled by the will of the patient. Exaggerated by emotions and tensions, they are accompanied by a particular mood.

Hemiballism

These are involuntary movements, without purpose, that are reminiscent of chorea, but are more violent, faster. They preferentially grab the proximal parts of the limbs and result from a combination of twisting, “throwing of the limbs,” pushing, shaking, and rotation movements. In general they are unilateral, but due to their intensity they move the entire body. They can respect the face and the trunk. They disappear with sleep.
At one time hemiballismus was attributed to lesions of the hypothalamic body of Luys, on the opposite side of the semiological manifestations. But hemiballism has been seen to appear due to lesions located outside the hypothalamic body of Luys.

Fibrillations and fasciculations

For a long time, both terms were confused or synonymous. With electromyography, we believe that the issue is clarified. We are going to comparatively summarize the characteristics of each of them.

Fibrillation. Not visible through the skin; independent and rhythmic contractions of individual muscle fibers, captured by electromyography. It occurs in denervated muscles, when the lesion is located in the anterior horns of the node.

Fasciculation. Visible through skin; contractile jerks of muscle sectors, brief, arrhythmic, limited to a muscle fascicle, incapable of producing movements. They represent the spontaneous contraction of a motor unit.

Although fasciculations can be found in degenerative processes of the anterior horn, as well as in root and trunk lesions, they do not therefore have a definitive topographic value of anterior horn injury and their study must be completed with the associated clinical and electromyographic findings.

Fasciculations include myomymias, undulating muscle contractions that can appear in tetany, uremia, thyrotoxicosis, nerve compressions, after muscular effort and tension states, being located, in the latter case, at the level of the orbicularis oculi and which is of great concern to patients. They are reminiscent of blepharospasm, but are milder, less intense and recur less frequently.

Seizures

Convulsions or convulsive attacks, manifested by muscle contractions that involve the entire body, can be included among abnormal movements, but they will be described in detail when discussing grand mal epileptic seizures. There we will see that, in complete cases, they can develop according to an always identical pattern for the same patient: aura, initial scream, tonic and clonic seizure phases, and postconvulsive manifestations.

Psychogenic hyperkinesias

Within these two groups can be included:

1. Of hysterical origin. In these hyperkinesias it is not possible to find any of the movements described above, but their imitation can be found. They are characterized by being singular, changing from time to time and influenced by suggestion.

   From the descriptive point of view of such movements, a collection of names has not yet been made, but the most frequent of them are tics and spasms. Many are compulsive and obsessive.

   They appear under the influence of an emotional affective factor. Some authors include "professional cramp" here, a fact highly discussed by many others. Those of clerks, violinists, etc. fall within professional cramps, and are manifested by the appearance of a muscle contraction, at the beginning or once the act has begun, which prevents its execution. While some may be psychogenic, there is no doubt that others are organic. We do not discuss this topic, which will be studied in Neurology treatises.
2. In mental patients. Alongside the functional abnormal movements, there are others. In this case there is the so-called parakinesia, in which there is a perversion of movement, which is strange, since the patient distorts it, mimicking a mixture of chorea, athetosis or psychomotor dyspraxia (forgetting the steps for the execution of a previously known movement), without there being paralysis or sensitivity disorders,

PARKINSONIAN SYNDROME

Concept

It is an extrapyramidal syndrome that is characterized by four cardinal signs: tremor at rest, bradykinesia, muscle rigidity and postural instability.

Syndramogenesis or pathophysiology

The most currently accepted pathophysiological mechanisms involved in the production of the symptoms and signs of this syndrome are:

1. Muscle rigidity: manifested by an increase in resistance against passive movement of a part of the body. It has been attributed to the disinhibition of the globus pallidus causing an increase in the suprasegmental activation of normal spinal reflex mechanisms.
2. Bradykinesia: manifested by slowness of automatic and spontaneous movements and impairment of the ability to initiate voluntary movements (akinesia).

   It has been proposed that this sign is due to the loss of the inhibitory action of dopamine in the striatum and the hypoactivity of the neurons in the external segment of the globus pallidus, which increases the tonic inhibitory stimulus of this basal nucleus. This would cause functional disinhibition (excitation) of the nucleus subthalamic and would cause an increase in neuronal activity of the internal segment of the pallidum, by elevating the tonic inhibitory stimulus from the globus pallidus to the thalamus and cortical projection areas. This alteration is evidenced by the increase in movement time that becomes prolonged when the patient performs sequential movements.

3. Tremor at rest (also called static): it is the rhythmic, more or less regular oscillation of a part of the body with respect to a fixed point with a frequency of 3-7 Hz, produced by alternating activity of agonist and antagonist muscles.

   Lesions of the striatum, the external region of the globus pallidus and the substantia nigra cause an increase in pallidofugal activity towards the thalamus, which causes tremor, as well as damage to the afferent systems that reach the thalamus from the cerebellum.

Syndromography or positive diagnosis of

parkinsonian tremor. In parkinsonian syndromes, a resting tremor occurs, which disappears with movement and affects the distal extremities of the limbs, particularly the hands (Fig. 54.3). This tremor is associated with muscle rigidity or hypertonia. In the hands it is observed as a continuous rubbing movement of the thumb with the index or with the other fingers, similar to the movement performed when counting coins.

When it affects the head, parkinsonian tremor is manifested by movements that resemble affirmation or denial; or by lip or mandibular movements.

Rigidity. Increased muscle tone that is distinguished from pyramidal hypertonia or spasticity in that it is not associated with hyperreflexia or Babinski sign. In the upper extremity it produces the cogwheel sign, which causes repeated passive flexions of the forearm on the arm. Extrapyramidal rigidity produces the sensation (when examining the patient) that there is a cogwheel mechanism in the elbow, which causes movement to occur in jerks and jerks.

Lack of facial expression. Mask facies.

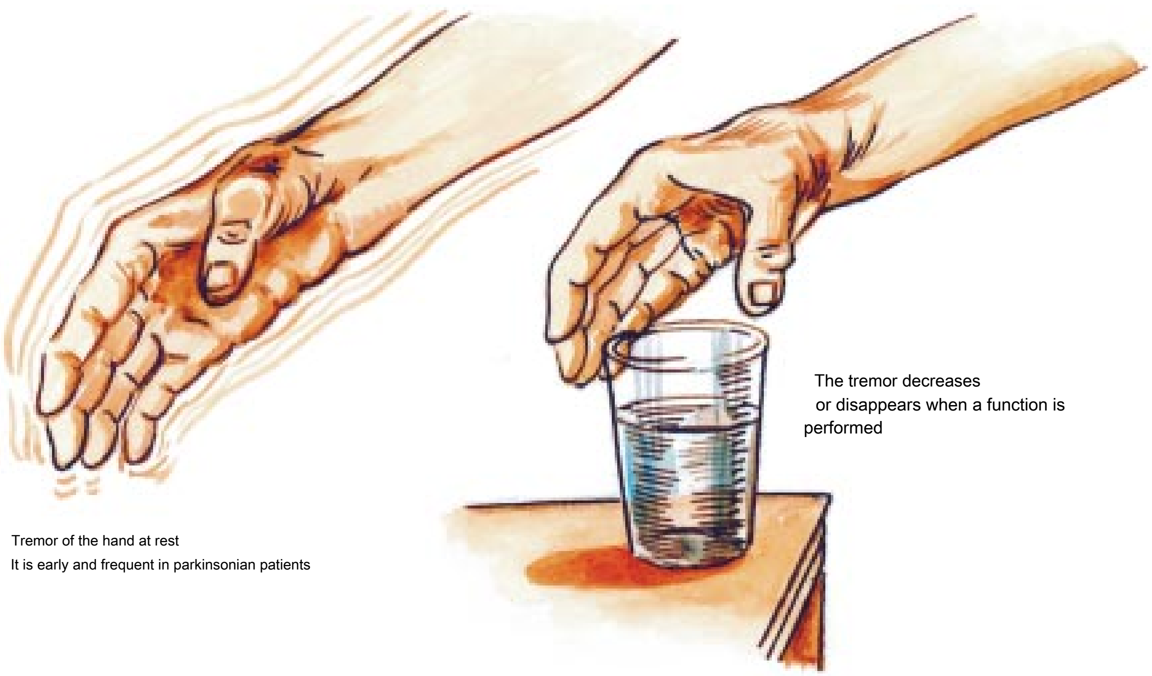
Parkinsonian attitude. Due to the predominance of the flexors, the patient appears still, with the head tilted and the trunk and extremities flexed.

Hypokinesia. Decrease in motor impulses.

March. It is in small steps, with insecurity, without associated movements of the arms; He walks as if leaning forward, the gait is unsteady and increasingly faster (the patient runs behind his center of gravity).

Maintained postures. They do not tolerate themselves well, they are changed frequently (akathisia).

Fig. 54.3 Parkinsonian tremor.



The tremor decreases
or disappears when a function is performed
Tremor of the hand at rest
It is early and frequent in parkinsonian patients

Etiology

The general practitioner must remember that Parkinson (an English neurologist) initially described the disease that bears his name under the name "paralysis agitans." Subsequently, numerous clinical conditions appeared that mimicked it, and according to our current knowledge we must remember that the following states may exist:

1. Parkinson's disease. Parenchymal disease (primitively affecting nerve cells), which is degenerative in nature and initially occurs in elderly people.

2. Parkinsonian syndromes. Under this name are the following tables:
   a) Postencephalitic or paraencephalitic Parkinsonism.
   It is of infectious origin and has a predilection for young people, although it does not respect age. The ointment face (seborrheic facies) and oculogyric crises are described as its own characteristics.
   b) Parkinsonism of vascular origin. In it we find pyramidal and extrapyramidal manifestations. In general, these are vascular, arteriosclerotic patients.
   c) Iatrogenic Parkinsonism. It is caused by the use of psychotropic drugs.
   d) Toxic Parkinsonism. It is due to carbon oxide, manganese, etc.
   e) Tumor Parkinsonism. It is advisable to be alert and not get carried away by the first impression of the syndrome and assign it another etiology.
   f) Traumatic Parkinsonism. This form has medicolegal importance, but is discussed by some authors.

CONVULSIVE SYNDROME

Concept

Convulsive syndrome is the name given to the set of clinical manifestations that have as their central element a type of involuntary movement called seizure.

Seizures are sudden, rhythmic jerks of large muscle groups.

The seizures come on suddenly; They are often accompanied by loss of consciousness, and their end is also more or less rapid.

There are two basic types of seizures:

1. The tonic seizure, in which the seizure is characterized by great rigidity or hypertonia, which can affect the entire body or parts of it.
2. The tonic-clonic seizure, in which there are alternating periods of tonic seizures and relaxation, which together produce the large alternating rhythmic movements of flexion and extension, especially of the limbs, that characterize this type of seizure.

Tonic-clonic seizures are the most common.

Syndromogenesis or physiopathology

Convulsive syndromes always occur due to hypersynchronous, very fast, high-voltage neuronal discharges, which can affect a neuronal group (focal or partial seizures) or the entire cerebral cortex (generalized seizures).

Electroencephalographic studies in grand mal seizures have made it possible to collect these neuronal discharges that appear in the form of large, pointed waves (spike). In the case of grand mal seizures and petit mal seizures (absences), the alterations appear at the same time in all regions of the cerebral cortex. This suggests that the origin of the discharge is not cortical, but in a point located deep and centrally in the brain. This area has been called the centerbrain and corresponds to the thalamic portion of the reticular substance (see coma). It is assumed that the discharge occurs in the highest part of the activating reticular system, reaching the cortex through the diffuse thalamic projection system and causing loss of consciousness; The same discharge, through the reticulobulbar and reticulospinal pathways, would cause seizures.

In the case of Jacksonian seizures, the origin is cortical, in the motor area or ascending frontal gyrus on the side opposite to that of the seizures. Crises of this type have great value in indicating the topography of the lesions. The site where they begin generally corresponds to the site of the cortex where the lesion is located (lower part of the ascending frontal gyrus on the opposite side in the case of facial seizures, upper part in the case of femoral seizures, middle zone in the case of brachial seizures).

The discharge (paroxysmal cerebral dysrhythmia) may remain localized in the cerebral cortex; but if it extends to the central brain, a grand mal crisis occurs.

This means that a grand mal seizure can originate primarily in the deep central region already described or in a cortico-subcortical focus, which is discharged first. This discharge corresponds clinically to the aura. That is why central brain epilepsies are called essential and do not have an aura; while epilepsies with aura are actually partial or focal epilepsies, with their origin in the cerebral cortex, which is secondarily they generalize. It is understood that the aura has localizing value and its character varies depending on the site of origin (visual in the occipital cortex, motor in the frontal cortex, sensory in the parietal lobe, etc.).

Syndromography or positive diagnosis

Although numerous classifications have been attempted in the clinical and electroencephalographic field, the following types are distinguished:

-Generalized tonic-clonic or grand mal seizure.

- Partial seizure, focal motor, Jacksonian or Bravais-Jackson.

- Status epilepticus.
- Tonic crises.

Generalized tonic-clonic or grand mal seizure 1.

Prodromes (precritical period). Hours before the start of the crisis, some patients report indefinite discomfort, sometimes headache, irritability.

apathy, tiredness, restless sleep; These symptoms that the patient or his family sometimes learn to detect as distant precursors to crises are called prodromes.

2. Critical period (beginning of the crisis). The grand mal crisis can begin in two ways:

a) The patient suddenly loses consciousness, falls to the ground as if struck by lightning, and the seizure begins immediately.

b) Seconds before the seizure begins and while the patient is still fully conscious, the aura or warning (motor, sensitive, psychic or sensory manifestation) is presented, which is always the same for each epileptic and which signals the beginning of the attack. In this case, the crisis does not completely surprise the patient, which sometimes allows him to avoid severe trauma, which frequently occurs in grand mal crises. In reality, it corresponds to the clinical manifestation of a focal crisis that generalizes, which is why it must be specified in the interrogation.

Visual auras: light or dark scotomas (vision of sparks, lights, dark or bright shapes that suddenly cross the visual field, etc.). Less frequently they can be complex figures (hallucinatory auras).

Auditory auras: ringing in the ears, voices, etc.

Olfactory (olfactory hallucination) and gustatory auras: These hallucinations are rare.

Sensitive auras: paresthesias, cold, etc.

Noticing auras: sometimes the patient presents a deviation of the head and eyes to one side (adversive crisis) or begins to have a localized tonic-clonic seizure (Bravais-Jackson type seizure) or raises an arm, as if in a defensive attitude (postural crisis). Vegetative auras:

palpitations, paleness, redness, abdominal pain, among others.

Others: more complex acts (scratching the nose, repeating a word, etc.).

When the grand mal crisis has an aura, this indicates that it has started in an area of the cerebral cortex (see pathophysiology).

3. Convulsive period of the crisis. As we defined, the patient falls to the ground not only suddenly, but also as if thrown or thrown by a great force; It does not fall softly, collapsing little by little, but with violence. This abruptness and violence of the fall explains the accidents that patients can suffer or cause: fractures, wounds, burns, car accidents, drowning, falls into space, and others; This will depend on the place and circumstances in which the crisis surprises you, the profession, etc.

a) Tonic phase: already on the ground, this is the first phase of the convulsive attack: the head backwards, the limbs extended, the jaws tightly clenched, the eyes fixed, the trunk in opisthotonos, the fingers flexed and the respiratory muscles completely paralyzed during expiration. Sometimes there is an initial scream, due to the sudden expiration (epileptic scream).

This phase lasts 10-30 seconds, and at the end of it the patient is often cyanotic.

b) Clonic phase, or better, tonic-clonic phase: it begins with the limbs; Alternating flexion-extension movements occur in the arms and legs, the convulsive access reaches the muscles of the face, which gives it an impressive, unpleasant appearance; the masseters suffer the convulsion, sometimes producing bites of the tongue; the eyes roll in their sockets; The respiratory muscles give up their contraction, breathing begins, at the beginning, in a jerky manner and at the end, in broader excursions, which causes the cyanosis of the tonic phase to disappear.

In this phase there is vegetative participation; there is sialorrhea, increased secretions of the respiratory tree, arterial hypertension, tachycardia, mydriasis, involuntary urination and, more rarely, ejaculation or defecation.

4. Postictal or postcritical period. After the crisis, the patient remains dejected, with great drowsiness. Sleep is of variable duration. When he wakes up, he often has a headache and muscle pain, and even a certain degree of confusion, but other patients wake up completely recovered. Rarely, a twilight state may occur, with mental confusion, disorientation and disorders in the relationship with the environment, which can last for hours. There is a capital fact: the patient does not remember anything about his crisis, with the sole exception of the aura, if it exists.

Partial seizure, focal motor, Jacksonian or Bravais-Jackson

It was described by Bravais in 1827 and later by Jackson (1861).

This is a tonic-clonic seizure that has two essential characteristics:

1. It is limited to one half of the body.
2. It is not accompanied by loss of consciousness. In this sense, we must clarify that a Jacksonian crisis can end up triggering a grand mal crisis, then the patient will lose consciousness and the focal crisis will give way to a generalized crisis.

The crisis generally begins in the arm (brachial type): the thumb is suddenly bent, the fingers are flexed, the forearm and arm are in a flexed position on the thorax; tonic-clonic seizures occur that progressively increase in intensity, then invade the neck and the corresponding hemiface, which appears convulsed and deviated towards the affected side;

The convulsive wave then extends to the lower limb, from the tip to the root. This extension, through the body, has been compared to an oil stain. In other cases the crisis remains localized in the arm. The crisis can begin with the face, spread to the upper limb and then to the lower limb (facial type). More rarely, it begins in the lower limb (crural type) and then spreads to the rest of the hemibody.

Sometimes, during the crisis, the patient cannot speak. At the end of the crisis (which lasts a few minutes) the seizures lose intensity and become more spaced out, until they finally disappear.

After the access, there may be paralysis in the affected body (Todd's paralysis) that disappears in a few hours. During this period, the presence of Babinski's sign on the affected side can be verified.

status epilepticus

When seizures of the grand mal type are repeated so close to each other that the patient between them If you do not regain consciousness, we speak of status epilepticus or status epilepticus, which is a serious complication that can end in death.

Jacksonian crises can also be repeated so closely that they produce a state of evil; It is 1continuous partial epilepsy (Kojevnikoff syndrome). It's strange.

Tonic crisis

Jackson described two types of tonic seizures with totally different prognostic significance, which are:

1. Tonic seizure of epileptic nature. It occurs especially in children and is characterized by a tonic contraction that can affect the face, causing a grimace (with open mouth, stretched lip corners), flexion of the upper limbs, extension of the lower limbs and contraction of the respiratory muscles, which cause a scream. But this crisis subsides, the child remains as before and then it may repeat itself again with the characteristic recurrence of the epileptic crisis.

2. Tonic crisis that is related to the decerebrate rigidity crisis. It always indicates a very serious condition and is produced by compression or injury to the brain stem. In these cases, in addition to the manifestations of the disease (generally a brain tumor or a stroke), tonic seizures occur in which the body contracts in extension, including the arms; These are presented in pronation, with closed hands; The tonic crisis determines a movement of the hands that is reminiscent of that of the cyclist when moving the ends of the bicycle handlebars.

   The head is also extended, the eyes look upward. There is a progressive alteration of the state of consciousness; There is alteration of pupillary light reflexes and respiratory and circulatory disorders. All of them attest to the extreme seriousness of these crises.

Complementary exams

In seizure syndromes, we fundamentally have:

1. Essential basic clinical laboratory tests.
2. Skull x-rays (simple, contrasted or both) or special.
3. Electroencephalogram of fundamental value to demonstrate the existence of a focus, its location and generally the type of epilepsy according to the characteristics of the waves.

Etiology

Convulsive syndromes can be due to the most diverse causes. These can be classified into four groups:

1. Gross, evolutionary lesions of the brain and its coverings. Brain tumor, epidural hematoma, subdural hematoma, cerebral embolisms, cerebral edema of any origin, meningoencephalitis, subarachnoid hemorrhage, brain metastases.

   It is accepted that some people are predisposed to seizures. This would explain why some patients (with the same injuries) have seizures and others do not.

2. Humoral disorders. That in some way affect the composition of the blood that nourishes the brain: hypoglycemia, hypocalcemia, cerebral anoxia of any cause, hyponatremia, intravascular coagulation, uremic coma, hepatic coma, exogenous toxins, etc.

3. Minimal, cortical, residual lesions (sequelae). Anoxia and perinatal trauma, post-traumatic scars, residual lesions of meningoencephalitis, sequelae of cerebral vascular accident, and others.

4. Crisis of unknown cause. In this group some congenital conditions and the so-called essential epilepsy can be found, in which a genetic factor is accepted; especially the group of central brain epilepsies (grand mal and petit mal).

It can be deduced that in the first two groups, seizures are usually another symptom of the disease that causes them; while in the remaining two, seizures (or others) constitute the main or only manifestation of the disease.

SENSORY SYNDROMES

Among the sensory syndromes we have:

- Sensory syndromes due to injury at various levels of the neuroaxis (they were studied in “Sensibility alterations”, Chapter 51).
-Headache.

HEADACHE

Concept

Since the time of Aretaeus of Cappadocia, a distinction has been made between cephalalgia (simple, ephemeral, throbbing headache) and cephalalgia (cranial discomfort in the form of heaviness, tension or simple sensation of presence, of existence of an area of the head; However, in practice these terms are used interchangeably.

Headache can constitute a syndrome in itself or be a symptom of other syndromes (intracranial hypertension, meningeal syndrome, arterial hypertension, etc.).

Syndromogenesis or pathophysiology

Headache presupposes the existence of nociceptive receptors that may be located in: scalp, epicranial aponeurosis, fascia, muscles, dura mater (in part), venous sinuses and veins, extracranial arteries and large-caliber cerebral arteries. It can also be caused by irritation of the V, VI, IX and X cranial nerves.

Tissues with little or no sensitivity are: leptomeninges, bones and brain parenchyma.

The mechanism of headache production is varied and we see that some causes act by:

1. Traction or displacement of sensitive structures (for example: brain tumors).
2. Direct irritation of these structures (meningitis, sinusitis).
3. Traction and dilation of the great intracranial vessels (cerebrospinal fluid hypotension after lumbar puncture).
4. Dilatation or distension of the intracranial or extracranial arteries (arterial hypertension).

Headaches in which the vascular factor is involved are called vascular headaches.

Classification

There are numerous classifications of headaches. We find it most useful that which distinguishes, according to their location in the clinic, two large groups:

1. Hemicranial or migraine headaches:
   a) Common migraine and ophthalmic migraine.
   b) Horton's histamine headache.
2. Non-migrainous headaches:
   a) Banal headaches due to mental or muscular tension.
   b) Symptomatic organic headaches.

In practice, the two most common headaches are: migraine and psychogenic tension headache.

Syndromography and etiology (rig. 54.4)

Some headaches have special characteristics, either because of the pain itself, or because of its prodromes or accompanying symptoms. We describe them below.

Migraine or migraine

It is a chronic recurrent headache that occurs due to attacks or attacks generally limited to one half of the head (hemicrania) related to sustained vasodilation of extracranial and intracranial arteries, and that occurs in predisposed people, either hereditarily or due to their personality.

Beginning. Generally half an hour or an hour before the onset of the headache, so-called auras (migraine warnings) may appear, which are non-painful and visual in nature:

Sparkling scotomas.

Dark scotomas.

Areas without vision in the visual field, with a hemianoptic distribution.

These phenomena are considered related to a brief initial period of arterial vasoconstriction.

Migraine crisis. The headache crisis then occurs, the intensity of which is lower at the beginning and then increases until it sometimes becomes unbearable. It is a unilateral headache, generally more intense towards the anterior region (retroorbital, frontal) that then extends to the temporoparietal region on the same side. The headache is stabbing and pulsating, that is, it is related to pulse waves. As commonly accentuated by light and noise, patients tend to go to bed in the dark. The pain, at first pulsatile and unilateral, can become constant and generalize to the entire head if the pain lasts several hours. This painful phase is considered to be produced by arterial vasodilation. There are frequently digestive manifestations: most patients lose their appetite and feel nauseated;

They may experience vomiting.

Other important data are:

Between attacks, the patient is headache-free.

The hemicrania changes location, that is, sometimes it is on the left side and other times on the right side. Migraines that are always on the same side should raise suspicion of an intracranial lesion.

Emotions can trigger crises; menstruation, alcohol, insomnia and dietary transgressions can also be triggering factors.

Histamine headache (cumulative headache, Horton syndrome)

It is also intense and hemicraneous, periodic and recurrent; but it is accompanied by tearing, rhinorrhea, nasal obstruction and ocular congestion on the same side as the pain.

Headache due to mental tension

Related to the muscular contraction maintained in the muscles of the neck and head, in relation to emotional tension, neighboring inflammations, cervicoarthrosis, etc.

Described with various characteristics by patients (tightness, stabbing, etc.), it is generalized to the entire head, although it can predominate in any region, without actually being a hemicrania; frequently in the occipital region and on the back of the neck.

Headache as a symptom of brain tumor

It is generally intense, diffuse, persistent, more marked when standing up; sometimes it is dull and increases with cough or exertion. It is accompanied by the rest of the elements of the intracranial hypertension syndrome. Other times, the tumor does not cause intracranial hypertension syndrome, but it is accompanied by headache. In these cases it can be caused directly by the tumor (it is unilateral or even more localized, sometimes over the site of the tumor).

Trigeminal neuralgia

It is unilateral and can affect any of the three branches of the nerve; It can occur spontaneously in the so-called essential neuralgia or be secondary to different factors in the so-called symptomatic neuralgia.

The pain becomes very painful and its location corresponds to the affected branch. Sometimes it is recurring.

Other causes that cause headaches

a) Visual diseases (glaucoma, refraction defects).

b) Sinusitis.

c) In meningeal syndrome (meningoencephalitis, haemorrhages).

d) High blood pressure crisis.

e) Infectious, toxic or medicinal processes.

f) Post-lumbar puncture headache.

BRAIN STEM SYNDROMES

As we have seen in volume 1, Chapters 14 and 15, the brain stem is a structure that includes the medulla or medulla oblongata, the pons or pons, and the cerebral peduncles; It will also have been seen that in the stem the long pathways that conduct motility (descending) and sensitivity (ascending) and the nuclei of the cranial nerves are closely related, in addition to other structures and numerous

A: ANGIOMA. Intracranial vascular malformations, such as angiomas, cause headaches due to distention of blood vessels and displacement of sensitive structures.

B: TUMORS. Intracranial neoplasms compress and displace sensitive formations, such as large arteries, veins, venous sinuses and some nerves, due to increased volume.

C: MENINGITIS. Traction of inflamed tissues, whose sensitivity to pain is increased, causes intense headache; The involvement of the roots of the upper cervical nerves and the structures of the posterior cranial fossa cause stiffness of the neck.

D: GLAUCOMA. The increase in intraocular pressure is capable of causing intense headache accompanied by nausea and vomiting; In some cases, headache is the initial symptom of this disorder.

E: SINUSITIS. Congestion and inflammation of the mucosa of the paranasal sinuses are a cause of referred pain, which is transmitted through the branches of the fifth cranial nerve.

F: 0STEOARTHRITIS. Osteoarthritis of the cervical spine causes contracture of the neck muscles, leading to headaches of variable frequency, intensity and location.

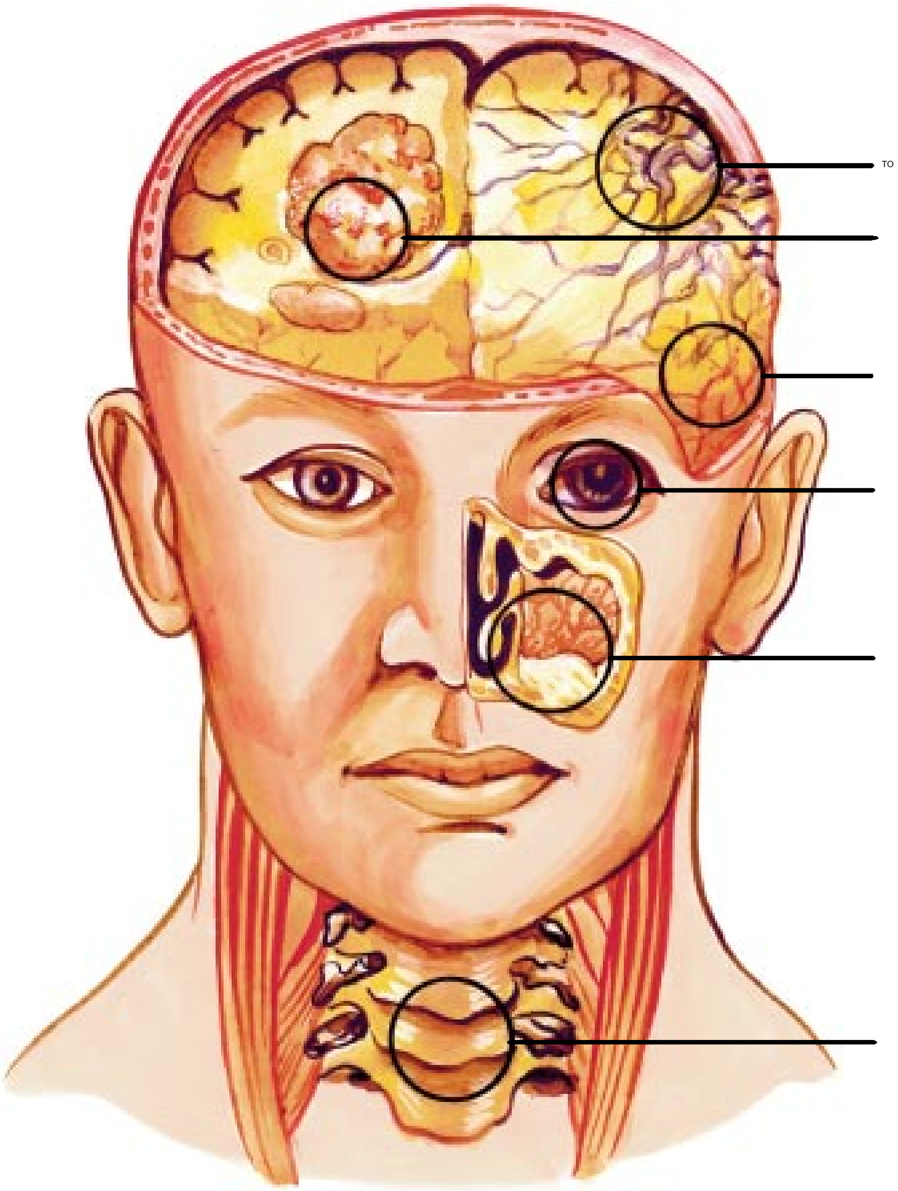


Fig. 54.4 Some of the common causes of headache.

pathways that connect the suprasegments with the spinal cord and its homologous formations in the stem itself.

This relationship occurs in a very small space, which explains why a small lesion can produce a very complex syndrome; Let us remember that the smallest drawings that we see on the structure of the stem (in the form of cuts) are five to ten times larger than the real size.

In this section we will refer to the main syndromes that these injuries can cause.

We have made reference to the concept of alternating hemiplegia, which is representative of brain stem syndromes; It includes injury to the pyramidal tract, on the opposite side (crossed pyramidal bundle) and injury to some cranial nerve, which manifests with symptoms on the same side. In this way, the different symptoms and signs allow us to specify the exact location of the lesions.

We will explain the main syndromes classically described, but we must understand that in such a small space, a small variation in the topography of the lesions can cause corresponding variations in the clinical components of these syndromes.

It includes:

Peduncular syndromes.

Pontine syndromes.

- Bulbar syndromes.

PEDUNCULAR SYNDROMES

Peduncular alternating hemiplegia (Weber syndrome) (see Fig. 51.4)

This syndrome is characterized by:

1. Direct and proportional total hemiplegia on the side opposite to the injury.
2. Eyelid ptosis, mydriasis on the same side as the lesion, and the eye deviating downward and outward.

This syndrome is produced by a lesion that affects the anterior part (foot) of the cerebral peduncle, through which the pyramidal bundle descends and at the same time the fibers from the nucleus of the I nerve, which emerge from the peduncle through this region.

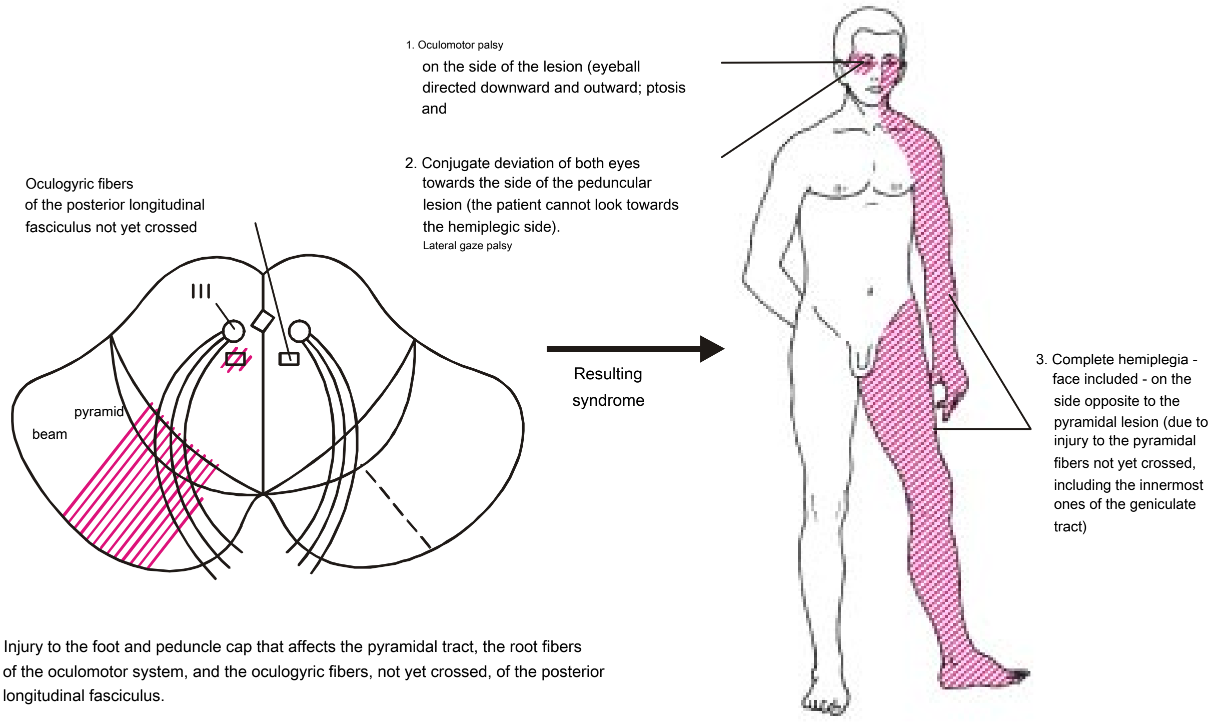
"Superior Foville syndrome or peduncular Foville" (rig. 54.5)

It is characterized by:

1. Weber syndrome as described.
2. Conjugate gaze paralysis (the patient looks towards the side of the lesion).

It corresponds to a lesion with a topography similar to the previous one, but which also affects the middle longitudinal fasciculus, also called the posterior longitudinal fasciculus (see functional anatomy of the III, IV and VI pairs).

Fig. 54.5 Superior Foville syndrome or alternating peduncular hemiplegia.


1. Oculomotor palsy
on the side of the lesion (eyeball directed downward and outward; ptosis and
2. Conjugate deviation of both eyes towards the side of the peduncular lesion (the patient cannot look towards the hemiplegic side).
Lateral gaze palsy
Oculogyric fibers of the posterior longitudinal fasciculus not yet crossed
III
pyramid
beam
Resulting syndrome
3. Complete hemiplegia - face included - on the side opposite to the pyramidal lesion (due to injury to the pyramidal fibers not yet crossed, including the innermost ones of the geniculate tract)

Injury to the foot and peduncle cap that affects the pyramidal tract, the root fibers of the oculomotor system, and the oculogyric fibers, not yet crossed, of the posterior longitudinal fasciculus.

Benedikt or posterior peduncular syndrome (fig. 54.6) This syndrome is characterized by:

1. Hemiasynergia, hemiataxia and hemihypotonia on the side opposite the injury,
2. Hemitetremor on the side opposite the injury.
3. Partial hemianesthesia on the side opposite the injury.
4. Hemiparesis on the side opposite the injury.
5. Oculomotor palsy on the side of the injury.

It corresponds to a lesion involving the superior cerebellar peduncle (opposite cerebellar hemisyndrome), the red nucleus (hemitetremor), the already crossed Reil ribbon (hemianesthesia) and the III nerve. As the lesion is posterior, the involvement of the pyramidal tract is partial.

PROTUBERANCIAL SYNDROMES

Anterior pontine syndrome (Millard-Gübler syndrome)

It is characterized by:

1. Hemiplegia that affects only the arm and leg on the opposite side of the injury.
2. Peripheral facial paralysis on the side of the injury.

It corresponds to a lesion in the anterior part of the pons, which affects the pyramidal bundle before the decussation, but having already detached the geniculate fasciculus that includes the fibers related to the nucleus of the VIth pair.

Inferior Foville syndrome or "Foville pontine" It is characterized by:

1. Millard-Gübler syndrome as described.
2. Conjugate gaze paralysis (the patient looks towards the opposite side of the lesion).

In this case, the lesion, in addition to affecting the pyramidal fibers and those of the facial nerve, affects the medial longitudinal fasciculus (already crossed), so the deviation of gaze is in the opposite direction to that of peduncular lesions.

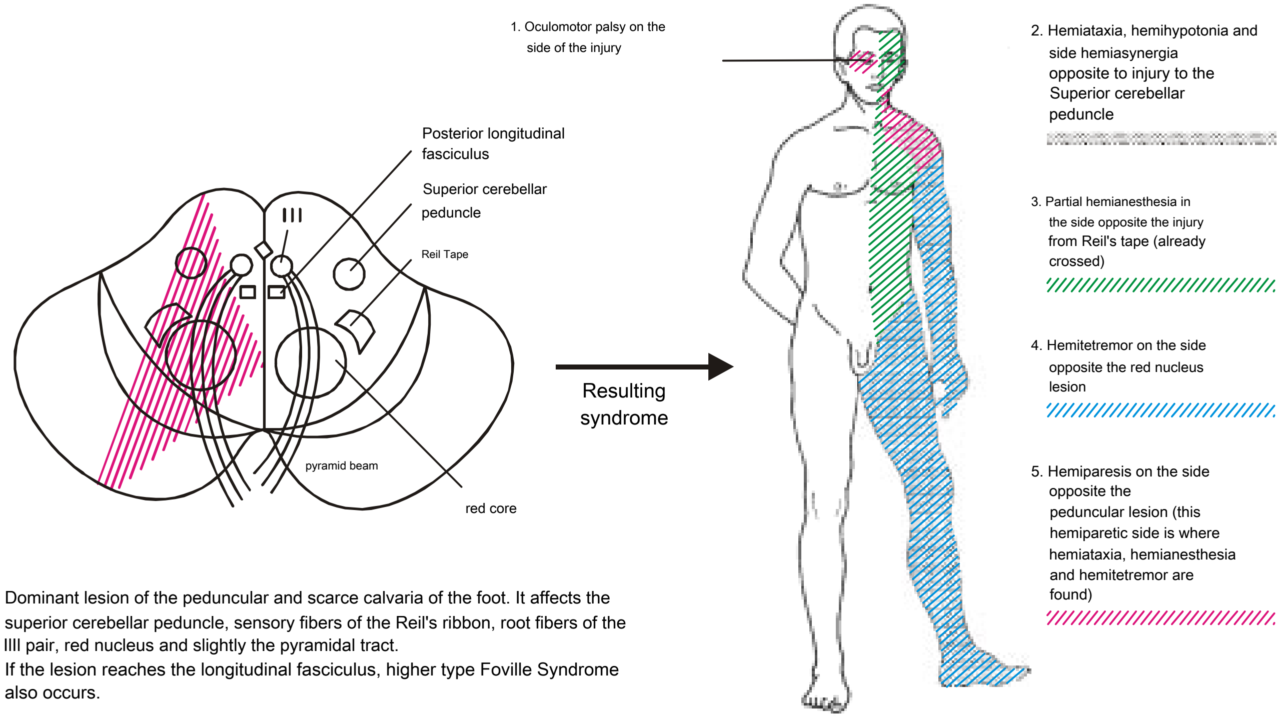
BULBAR SYNDROMES

Lateral bulbar, Wallenberg, or posterior and inferior cerebellar artery syndrome

It is characterized by:

1. Alternating thermoanalgesic hemianesthesia at the level of the limbs on the side opposite to the injury, while in the face there is paresthesia or anesthesia on the side of the injury.
2. Hemianesthesia of the face on the same side as the injury.
3. Pharyngolaryngeal and velopalatine paralysis on the same side as the lesion.

Fig. 54.6 Benedikt syndrome or posterior peduncular alternating hemiplegia.


1. Oculomotor palsy on the side of the injury
Posterior longitudinal fasciculus
Superior cerebellar peduncle
Reil Tape
III
pyramid beam
red core
Resulting syndrome
2. Hemiataxia, hemihypotonia and side hemiasynergia opposite to injury to the Superior cerebellar peduncle
3. Partial hemianesthesia in the side opposite the injury from Reil's tape (already crossed)
4. Hemitetremor on the side opposite the red nucleus lesion
5. Hemiparesis on the side opposite the peduncular lesion (this hemiparetic side is where hemiataxia, hemianesthesia and hemitetremor are found)

Dominant lesion of the peduncular and scarce calvaria of the foot. It affects the superior cerebellar peduncle, sensory fibers of the Reil's ribbon, root fibers of the IIII pair, red nucleus and slightly the pyramidal tract.
If the lesion reaches the longitudinal fasciculus, higher type Foville Syndrome also occurs.

4. Hemiasynergia and lateropulsion on the same side of the injury.
5. Paralysis with atrophy of the half of the tongue corresponding to the side of the lesion.
6. Claude-Horner syndrome on the same side as the lesion.

Its most common cause is occlusion of the posterior inferior cerebellar artery. As the posterior and lateral regions of the bulb are affected, the following structures are affected, which express the symptoms and signs:

1. Lateral spinothalamic bundle, which conducts thermoalgesic sensitivity and has already crossed into the different spinal segments.
2. Spinal trigeminal nucleus.
3. Ambiguous nucleus.
4. Inferior cerebellar peduncle (restiform body).
5. Nucleus of the hypoglossus.
6. Oculosympathetic fibers, which descend through the medulla from the hypothalamus to reach the Budge ciliospinal center.

Anterior bulbar syndrome

It is characterized by:

1. Hemiplegia that does not affect the face on the opposite side of the injury.
2. Paralysis of the hypoglossus on the same side as the lesion (lingual hemiatrophy, deviation of the tip of the tongue towards the side of the hemiatrophy).

It corresponds to an injury to the bulbar pyramids before their crossing, with injury to the fibers coming from the hypoglossal nucleus.

Posterior bulbar syndromes

They are classically described:

1. Avellis syndrome. Characterized by contralateral hemiplegia that does not affect the face and paralysis of the ipsilateral vocal cord and soft palate.
2. It corresponds to a lesion of the pyramidal pathway not yet crossed and of the fibers coming from the nucleus ambiguus.
3. Schmidt syndrome. It is a contralateral bulbar type hemiplegia (does not affect the face), with paralysis of the sternocleidomastoid and ipsilateral trapezius, due to injury to the XI nerve.
4. Jackson syndrome. Velopalatopharyngolaryngeal hemniplegia associated with paralysis of the sternocleidomastoid muscles, the trapezius and half of the tongue, all on the same side as the hemiplegic lesion of the arm and leg on the opposite side that does not affect the Face.

CEREBELLAR SYNDROME

Concept

The set of symptoms and signs caused by various injuries or disorders that affect the cerebellum or its pathways is called cerebellar syndrome.

General considerations

1. Primitively the cerebellum is not an organ of motor function, since from a phylogenetic point of view it develops outside the primary vestibular area, but it is closely associated with the vestibular system and proprioceptive impulses (called by Déjerine unconscious deep sensitivity).
2. Within the zoological scale it reaches a certain level of development from birds, in which it plays an important role in balance and orientation.
   But it reaches its greatest evolution in mammals, which have coordinated movements in their extremities.
3. Its development moves in step with the cerebral hemispheres and is closely related to the pyramidal and extrapyramidal motor components and the reticular substance of the brain stem, playing an important role in the control and integration of its motor function.

Anatomophysiological count

There is a purely morphological description of the cerebellum, which divides it into a vermis and two lateral hemispheres. This will be the one that we will use to detail the semiological disorders, whether general or topographical. Such a morphological description does not respond to phylogenetic knowledge, which is quite complex for the student and the general practitioner.

According to the phylogenetic period of appearance, three parts have been described: archicerebellum (or center of vestibular balance); paleocerebellum, which ensures postural tone, that of muscles in general, including the extrinsic muscles of the eyes, as well as the control of vegetative functions, and neocerebellum, responsible for the automatic control of voluntary motility.

Archicerebellum

1. Afferent pathways. They are all subcortical and arise in the acoustic spots of the utricle, saccule and acoustic ridges of the semicircular canals. The sensory protoneuron is located in Scarpa's ganglion, and from there, via the vestibular nerve, it sends its cylinder axes to the bulbopontine vestibular nuclei.
   From the vestibular nuclei, a second neuron carries

the influences to the flocculus via the inferior cerebellar peduncle.

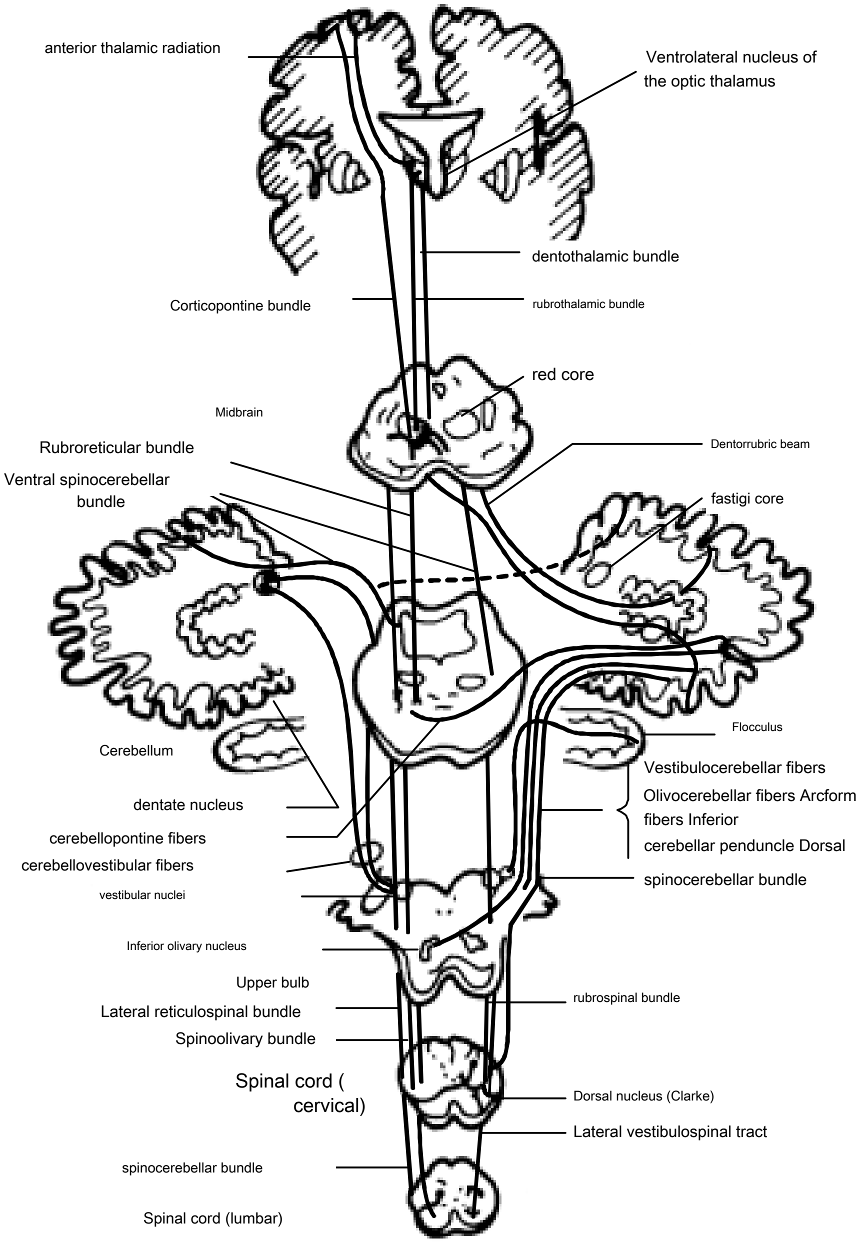
2. Efferent pathways. There is a rule that says that the cerebellar pathways that start from the cerebellar cortex always make a stop, a relay, in one of the central gray nuclei of the cerebellum. From the cortex of the flocculus they pass to the dentate nucleus of the cerebellum, from where a second pathway sends fibers that return to the vestibular nuclei. A third neuron, a crossed vestibulospinal neuron, originates from these nuclei and goes to the anterior horn of the spinal cord (Fig. 54.7).

Paleocerebellum

1. Afferent pathways. It carries unconscious proprioceptive sensations through the cerebellar fascicles, direct (Flechsig) and crossed (Gowers), which end in the central lobe and the culmen. Other elements are added from the bulb: fibers from the Von Monakow nucleus, fibers of olivary origin and from the bulbopontine sensory nuclei.

2. Efferent pathways. After a stop at the cores of the roof and incidentally in the olives, two routes follow: the first, crossed, which goes to the anti-

Fig. 54.7 Main afferent and efferent connections of the cerebellum.


anterior thalamic radiation
Ventrolateral nucleus of the optic thalamus
dentothalamic bundle
Corticopontine bundle
rubrothalamic bundle
red core
Midbrain
Rubroreticular bundle
Dentorrubric beam
Ventral spinocerebellar bundle
fastigi core
Cerebellum
Flocculus
Vestibulocerebellar fibers
Olivocerebellar fibers Arcform fibers Inferior cerebellar penduncle Dorsal
spinocerebellar bundle
dentate nucleus
cerebellopontine fibers
cerebellovestibular fibers
vestibular nuclei
Inferior olivary nucleus
Upper bulb
rubrospinal bundle
Lateral reticulospinal bundle
Spinoolivary bundle
Spinal cord (cervical)
Dorsal nucleus (Clarke)
Lateral vestibulospinal tract
spinocerebellar bundle
Spinal cord (lumbar)

guide to the red nucleus (magnocellular), from which the rubrospinal tract emerges; A second contingent, ipsilateral, through the inferior cerebellar peduncle goes to the vestibular nuclei: it is the vestibulospinal tract.

The connections of the rubrospinal tract with the extrinsic motor nuclei of the eye explain the action on their tone.

The two circuits, paleocerobellar and archicerebellar, are subcortical (with reference to the cerebral cortex); consequently, they are unconscious.

Neocerebellum

1. Afferent pathways. Its path is made up of two neurons. The first is the cortico (cerebral)-pontine, born in the second temporal gyrus (known as temporopontine), crosses the sublenticular region and occupies the external fifth of the foot of the cerebral peduncle to end in the middle of the transverse fibers of the pons. At the level of the nuclei of the pons it articulates with the second neuron (pontocerebellar), which goes to the neocerebellum (cortex), is located behind the primary sulcus and reaches the posterior part of the lateral lobe (ansiform and semilunar lobe).

2. Efferent pathways. They are made up of numerous neurons. They go from the cerebellar cortex to the dentate nucleus, from this last nucleus they go to the optic thalamus, after crossing over (Wernekinck's).
   Finally, from the optic thalamus they go to the second temporal gyrus (from where the temporopontine pathway departed) and they also go to the prerolandic gyrus, from where they will depart to their final term, the main motor or corticospinal pathway. For some, it is possible that this cerebellar circuit is not incorporated into the pyramidal pathway, but into the extrapyramidal pathway (adversive fibers, Delmas and Delmas), responsible for ensuring the coordination of semi-automatic and semi-voluntary movements.

In summary, the useful data for the clinician are the following: the cerebellum is composed of a medial part, narrow, elongated from front to back, which is the oldest phylogenetically (vermis), and of two lateral and voluminous lobes, the cerebellar hemispheres, whose phylogenetic development is parallel to that of the forelimbs and hindlimbs, in terms of their ability to carry out independent activities.

We distinguish:

1. A posteroinferior, medial and paramedial part, which is the oldest (archicerebellum), whose connections are vestibular, and which is involved in maintaining balance.
2. A middle and anterior, medialy and paramedial, more recent part (paleocerebellum), which modulates the activity of the muscles of the body axis and. therefore, the tone and movements in the upright station and in walking.
3. Two lateral portions, which constitute the majority of the hemispheres, which are the most recent (neocerebellum), and which control the independent motor activity of the limbs.

The organization of the afferent and efferent pathways of the cerebellum is such that the disorders are ipsilateral to the lesions. The only exception to this rule are lesions located between the decussation of the superior cerebellar peduncles and the red nucleus.

Cerebellar regulation of motility, to a large extent, is carried out through the gamma system. Essentially, lesions of the cerebellum determine: a decrease in the myotatic reflex (stretch reflex or traction reflex) and an alteration in the temporal concordance of the activity of the agonist-antagonist-synergist muscle groups, whose coordinating action conditions normal harmonious movement. These fundamental disorders have two main clinical consequences: muscular hypotonia and movement dysmetria, or lack of continuity of the activities of the muscle groups during the movement, which determines incoordination.

Syndromography or positive diagnosis

Clinical picture

1. Cerebellar ataxia. It is the capital disorder of the syndrome; It is both a static and dynamic ataxia. In the standing posture, wide and continuous oscillations of the trunk are observed, which are also accompanied by staggering, in relation to excessive, alternating contractions of the leg muscles; Despite this, the patient does not fall if he closes his eyes, so the Romberg sign is negative. During walking, dynamic ataxia is checked: cerebellar gait.

   We will remember that it is a hesitant walk, like that of a drunk; the base of support is expanded (legs separated), and the arms are open; The walk is carried out in a zigzag, with frequent deviations to one side or the other, as if the patient were going to fall; takes disproportionate steps and stops and starts late; But despite the insecurity of the walk, he almost never falls, and the disorder does not increase when closing his eyes, in terms of the decomposition of the movement, but it does increase in terms of the deviation of the direction of the walk. The star gait is characterized by this systematic deviation towards the same side.
2. Dysmetria. Movement, even the most basic, is exaggerated, out of measure. It is explored with the index-nose and heel-knee tests (see Fig. 14.4, volume 1).

3. Asynergy. The movements complexes HE decompose, they are not coordinated in time. In the normal subject, if the head and trunk are tilted backwards, a synergistic flexion of the feet and knees occurs to avoid falling. In the cerebellar patient, when making the same backward inclination, the legs remain extended and if they are not supported, they fall (fig. 54.8).

4.Adiadochokinesia or dysdiadochokinesia. Successive movements such as pronation and supination of the hands (puppet test) cannot be performed quickly.

5.Dyschronometry. Delay in the beginning and end of movements.

6.Tremor. The cerebellar tremor is extensive and intentional character. It is an expression of the incoordination of voluntary movement that causes its decomposition, observing marked oscillations.

falls.

7.Hypotonia. In the cerebellar patient there is hypotonia and increased passive motility. To demonstrate this passivity, several tests are used.

The Holmes-Stewart or rebound test consists of inviting the patient to flex the forearm on the arm, while the examiner opposes said movement; In a normal subject, when the forearm is suddenly released it is directed towards the shoulder, but returns forward due to the tone of the antagonists; In the cerebellar patient, the hand collides strongly with the shoulder (Fig. 54.9).

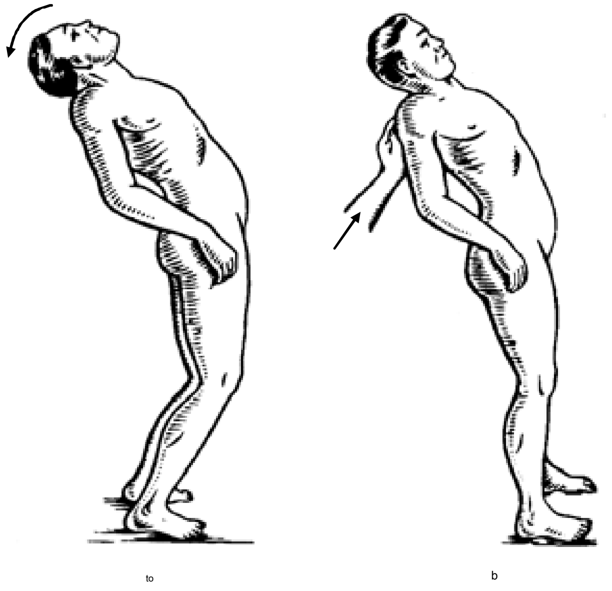


Fig. 54.8 Cerebellar asynergia: a, the normal individual, when tilting the head and trunk backwards, flexes the feet and knees to compensate for the inclination and not fall; b, in the cerebellar patient these compensatory synergistic movements stop taking place and when he leans his head back, he collapses if he is not supported.

8. Language disorders. The word is monotonous, lacks modulation, scanned (slow, syllabled) and explosive.

9. Nystagmus. It is not frequent. It occurs when the archicerebellum is

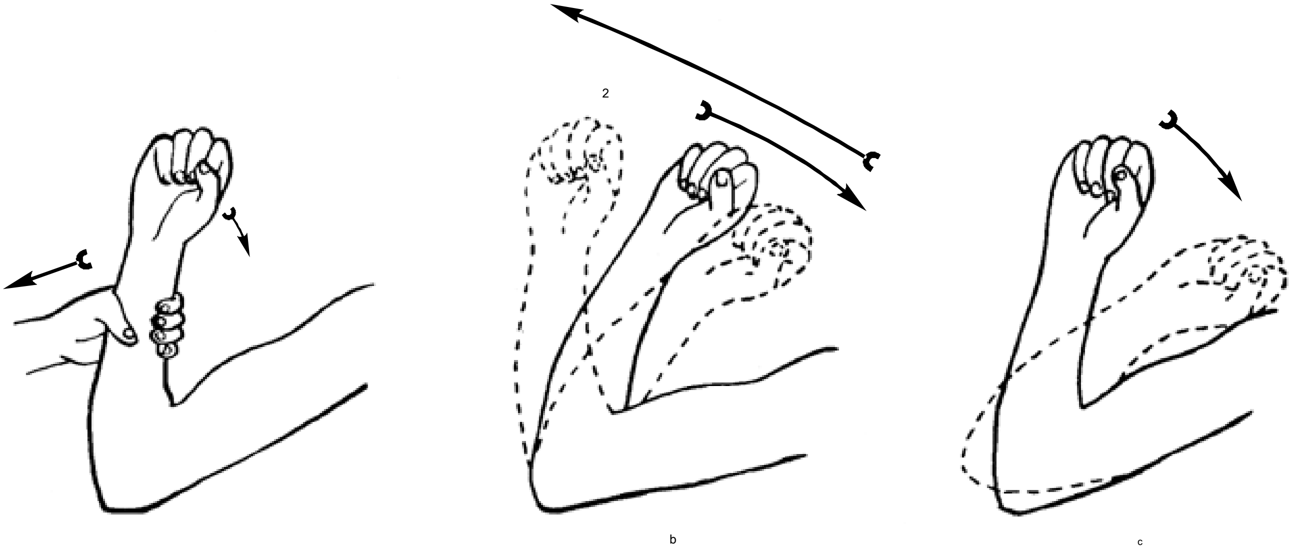


Fig. 54.9 Holmes-Stewart or rebound test: a, the doctor opposes flexion; b, the doctor releases the forearm: flexion continues, but stops at 1, to extend again at 2 (normal test); c, when the doctor releases the forearm, the flexion continues until the forearm makes contact with the shoulder (response in the cerebellar patient).

Topography of cerebellar syndrome

Depending on the topography of the lesions, variants of the syndrome may be observed.

Cerebellar vermis syndrome0 0 static0

a) The paleocerebellum is affected.
b) Corresponds to lesions of the vermis.
c) It is characterized by static instability and gait disorders.

The aforementioned disorders are marked in the trunk and lower limbs.

Hemispheres or kinetic syndrome

a) The neocerebellum is affected.
b) It is characterized by: dysmetria, asynergia, adiadokinesia, dyschronometry, tremor and
c) If the hemispheric injury is unilateral, the symptoms will occur on the same side as the injury.

Mixed syndrome

It is caused by injury to the vermis and hemispheres.
In this syndrome, the symptoms of the previous two overlap.

Etiology

1. Vascular syndromes: cerebellar hemorrhage, cerebellar infarction.
2. Tumor syndromes: cerebellar tumors, aneurysms that compress the III nerve.
3. Heredodegenerative conditions: Friedreich's disease, Pierre-Marie's cerebellar heredoataxia, and others.
4. Demyelinating diseases.
5. Infectious syndromes: abscess, tuberculoma.
6. Other causes: poisoning (manganese, organic mercurials), reversible toxicity due to diphenylhydantoin, alcohol, myxedema, plaque sclerosis.

MENINGEAL SYNDROME

Concept

The clinical condition caused by the invasion or acute aggression of the cerebrospinal meninges as a consequence of some pathological process, generally infectious, is called meningeal syndrome.

There are many factors capable of causing a meningeal syndrome, among which we highlight infectious, toxic and those linked to vascular processes, such as meningeal hemorrhages due to rupture of congenital or acquired aneurysms.

Many times it is not a purely meningeal problem, but rather a brain condition is associated with it, thus forming meningoencephalitis. We remember that we are talking about meningeal syndrome in general and not meningitis, since to make the latter diagnosis a lumbar puncture is mandatory in order to demonstrate the existence of inflammatory alterations at cerebrospinal fluid level.

Within meningeal syndrome, the general practitioner must distinguish the following circumstances: meningitis, when infectious-type alterations in the cerebrospinal fluid are added to the clinical picture; meningeal reaction, which can occur after an aseptic lumbar puncture or after pneumoencephalography (injection of air into the subarachnoid space), and in which very slight alterations in the cerebrospinal fluid are added to the clinical picture; meningism, where we only find a very slight clinical picture and the lumbar puncture produces a normal fluid in the laboratory tests and meningeal hemorrhage, in which bloody, or xanthochromic, cerebrospinal fluid is added to the clinical picture.

Brief anatomical account

The meninges form a membranous envelope that separates the nervous substance from the craniospinal cavity where it is contained.

Three meningeal membranes are described: dura mater, arachnoid mater and pia mater. The arachnoid is in turn formed by two leaves, limiting between them a virtual cavity, the arachnoid cavity, while the deep or visceral leaf of the arachnoid is separated from the pia mater by the subarachnoid spaces. In practical terms, we consider the dura mater as the fibrous membrane attached to the bone. The arachnoid, like the membrane that lines the deep side of the dura mater. The pia mater, which covers the nervous tissue.

Between the arachnoid and the pia mater are the subarachnoid spaces through which the cerebrospinal fluid circulates. These spaces communicate with the ependymoventricular cavities through the foramina of Luschka and Magendie. Cerebrospinal fluid (CSF) has been studied in Chapter 53.

When there is a meningeal reaction accompanied by hyperproduction of CSF, an increase in intracranial pressure results, leading to intracranial hypertension syndrome (headache, vomiting); lesions of the underlying cerebral cortex (parasis of ocular nerves, excitement phenomena, convulsions, monoplegia, etc.); irritation of the spinal roots (contractures) that affect muscles of the neck, spine, and lower limbs and that fix the patient in positions that prevent the stretching of the inflamed spinal roots (gun trigger attitude, neck stiffness, and others).

Syndromography or positive diagnosis

Cynical picture

Functional symptoms

The so-called meningeal triad is described: headache, vomiting and constipation.

1. Headache. Constant or remitting, very intense to the point of eliciting moans or screams from the patient ("meningitic cry" of the child); It is exaggerated by light, noise, active or passive movements and efforts (coughing, sneezing).
2. Vomiting. Central type, easy, effortless, in jet; They are caused by changes in position.
3. Constipation. It has less semiological value.

Physical examination

muscle contracture.

It is a constant sign and its verification is enough to guide the diagnosis. It's precocious.

Trunk contracture can reach a pronounced degree, causing opisthotonos; other times the subject is in a position called rifle trigger.

These positions are, for some authors, antalgic, as we have pointed out in pathophysiology.

Other times, however, the picture is overlapping and then a series of maneuvers are useful to reveal the painful muscle contracture, which must be carried out delicately, since they cause severe pain.

The so-called neck stiffness due to muscle contracture is first investigated; Pass your hand under the subject's head and try lifting the body through this support point; Normally the head flexes under the pressure of the hand, if there is rigidity, the head remains in extension and the subject manifests pain.

The muscle contracture also affects the trunk and lower limbs. It is investigated using the Kernig and Brudzinski signs (see volume 1, Chapter 14).

Other symptoms and signs

1. Seizures. Frequent and widespread, especially in children and in meningoencephalitis of the convexity of the brain.
2. Paralysis. Incomplete, mobile and fleeting.
3. Sensory disorders. Photophobia frequently: there may be hyperacusis.
4. Cutaneous hyperesthesia. It is frequent.
5. Mental disorders.
6. Vasomotor disorders. Trousseau's meningitic line, that is, the persistence of a red line long after passing a blunt object over the patient's abdomen or thigh, has no senmiological value and only indicates capillary disorders.
7. General signs. Fever is common and ranges between 39-40 °C.

Like any infectious syndrome, there is a coated tongue, thirst, oliguria, and anorexia.

Complementary exams

1. Lumbar puncture confirms the diagnosis. The liquid may have the following characters:
   a) Clear as rice water: viral and tuberculous meningoencephalitis (the latter already very rare in Cuba)
   b) Purulent turbidity: bacterial meningoencephalitis.
   c) Hemorrhagic: subarachnoid hemorrhage.
   d) Xanthochromic0: subarachnoid or intracerebral hemorrhage.
2. The pressure is increased, it can reach up to 50-100 cm of water.
3. Albumins are increased.
4. Glucose decreases in acute bacterial meningitis.
5. Chlorides decrease slightly but their decrease is greater in tuberculosis.
6. There is an increase in the number of cells, which can reach 3, 000 and 4,000 elements or more; In bacterial meningoencephalitis, the increase is mainly in polynuclear cells, and in viral cases, in elements. lymphocytes.
7. The etiological diagnosis is made by culture of said fluid (meningococcus, pneumococcus, Koch's bacilli, Eberth's, etc.).
8. Colloidal reactions give precipitation in the tubes located on the right side that is characteristic of meningitis.

Etiology

The most common cause of meningeal syndrome is meningoencephalitis, which can be caused by: viruses, bacteria (meningococcus, pneumococcus, staphylococcus, gram-negative bacilli and tubercle bacillus), rickettsiae, fungi, spirochetes (leptospirosis, syphilis) and protozoans (Laverán's haematozoan). In these cases we speak of primary meningoencephalitis, because these agents directly attack the central nervous system.

When the etiological agent does not primarily attack the brain and the allergic or hypersensitivity factor is the fundamental mechanism, the term secondary meningoencephalitis is used. Representations of this group are those seen in measles, chickenpox and post-vaccinations (rabies, smallpox, etc.).

Subarachnoid hemorrhages can also cause a meningeal syndrome, but in these cases the syndrome is never as evident as in meningoencephalitis. Fever appears 24-48 hours after the onset of symptoms, and is never as high as it can be in bacterial meningoencephalitis.

ENDOCRANIAL HYPERTENSION SYNDROME

Concept

Intracranial hypertension is the increase in the pressure of the cerebrospinal fluid within the skull cavity. This syndrome is characterized by a set of subjective or objective manifestations (or both at the same time) that result from the disturbance in the balance that normally exists between the braincase (continent) and the brain (contents), including the vessels and CSF in the latter.

Intracranial hypertension is manifested by a series of symptoms whose presence allows its diagnosis. The main symptomatic triad is: headache, vomiting and optic papilla edema.

Syndromogenesis or pathophysiology

Intracranial hypertension can have several production mechanisms:

1. The skull is an inextensible cavity, therefore a lesion of a certain size, such as a tumor, when it grows, occupies a space that displaces or compresses the brain structures contained therein. This mechanism has some importance in tumors of a certain volume and rapid growth, such as subdural or epidural hematomas.

2. But the most important production mechanism is the obstacle to the free circulation of CSF. As is known, this is produced by the choroid plexuses of the cerebral ventricles, circulates to the third ventricle through the foramina of Monro, passes to the fourth through the aqueduct of Sylvius and exits towards the subarachnoid space (where it is reabsorbed) through the foramina that connect the fourth ventricle with this space. A small tumor that prevents, for example, the exit of CSF to the Sylvian aqueduct, will produce great intracranial hypertension. On the other hand, a frontal tumor can grow until it reaches a certain volume without hindering CSF circulation and, consequently, without producing intracranial hypertension.

3. Another mechanism by which intracranial hypertension can occur is cerebral edema. It is important in the genesis of hypertension intracranial disease that can accompany meningoencephalitis or hypertensive

4. In subarachnoid hemorrhage, a large amount of bleeding occurs in the aforementioned space, which, due to its rapidity, exceeds the physiological possibilities of reabsorption, which causes intracranial hypertension. Also in meningoencephalitis there may be excess CSF production.

Severe intracranial hypertension produces pressure cones through two sites: the foramen of the tentorium cerebellum, through which the brain stem passes (supratentorial tumors) and the foramen occipital (posterior fossa tumors).

These pressure cones can produce wedge at the peduncular and bulbar level, which constitutes a serious complication of intracranial hypertension; They also cause hernias of the temporal lobe, cerebellum, etc.

Syndromography or positive diagnosis

Clinical picture

It usually presents a triad of manifestations to which the greatest attention must be paid, since any of its three members "can open the scene." The triad is made up of the following elements:

1. Headache. It is intense, generally diffuse, it can be constant or intermittent; Its production mechanism is due to the distention of the meninges due to hypertensive CSF or the traction of sensitive structures. It can be so intense that the patient is prevented from moving and refuses to get out of bed or even feed himself: but sometimes its intensity is not marked.

   The headache is increased by exertion (coughing and sneezing, especially). It may have a specific location in certain tumors of the occipital region.

   Example: cerebellar tumors.

2. Vomiting. They are due to the compression that hypertension exerts on the medulla oblongata and the excitation of its centers. They are not preceded by nausea, they are sudden and in a gush or burst. On many occasions they do not present in this typical form since the individual has eaten food and there is nausea preceding vomiting.

3. Edema of the papilla. This symptom can lead to consecutive atrophy of the optic papilla and is related to the increase in intracranial pressure communicated to the subarachnoid space that surrounds the optic nerve and retinal vessels. The effects of edema are threefold: effacement and elevation of the edge of the papilla, venous engorgement, and narrowing of the retinal arteries. Papilledema is the pathognomonic sign of intracranial hypertension, even though it is absent in twenty percent or more of cases.

4. Other symptoms are: constipation and bradycardia, produced by the same mechanism as vomiting; drowsiness and psychological alterations caused by cerebral edema. In all well-developed intracranial hypertension, dullness or psychic slowing; Seizures and vertigo may also occur.

Symptoms and signs depending on the cause and location

The symptoms and signs already described of intracranial hypertension, depending on their cause, may be accompanied by other symptoms and signs that are not due to CSF hypertension, but to the lesion originating from the syndrome and its particular topography.

It should be clarified that intracranial hypertension can produce false localization signs, the most important of which is the palsy of the VI cranial nerve.

Let's now see the topography and the signs that correspond to it:

1. Supratentorial tumors:

a) Frontal tumors. As already said, in the anterior part of the lobe a tumor can grow until it reaches a considerable volume without producing great intracranial hypertension. They are manifested by mental disorders (loss of interest in cleanliness, family, work), childish euphoria (death), disorders of judgment and memory.

They can also produce ataxia and forced grip reflex. This reflex is explored by stimulating the palm of the patient's hand with a finger, who will involuntarily press said finger. It is valuable, above all, when it is unilateral and on the opposite side of the injury. Sometimes it is accompanied by an uncontrollable tendency of the patient to take all objects with his hand, without a precise objective.

Tumors of the inferior and anterior face of the frontal lobe can cause anosmia on the side of the lesion due to compression of the olfactory tract or amaurosis due to compression of the optic nerve. Which can lead to optic atrophy. In these cases, the fundus examination detects optic atrophy on the side of the tumor and papilledema in the other eye due to intracranial hypertension (Foster-Kennedy syndrome).

If the tumor is located in the ascending frontal gyrus, it will produce focal motor epileptic seizures on the side opposite the lesion. They are called Jacksonian seizures or Bravai-Jackson seizures (see "Seizure syndrome" in this chapter).
The aforementioned crises, an expression of irritation of the motor region, may be replaced by paralysis. (hemiplegia or monoplegia) that will affect the opposite hemibody, since it expresses destruction of the motor region.

b) Parietal tumors. The most important sign is stereognosia disorder. They can also cause hemianopias in quadrants (quadrantopsias) and sensory aphasias, the latter if the lesion is located in the posterior region of the parietal lobe.

c)Temporary tumors. They can cause psychomotor or other temporal lobe epileptic seizures (uncinate, hallucinatory, psychic seizures, etc.).
They can also cause aphasia and hemianopsia.
For language disorders to exist (aphasias or dysphasias), they must affect the dominant hemisphere.

d) Occipital tumors. Visual hallucinations and quadrantpsias occur. Sometimes they give cerebellar symptoms by compressing this neighboring structure.

e) Tumors of the third ventricle. They only cause symptoms of intracranial hypertension, which can take on two characteristics: intense (the most common) or intermittent.

f) Pituitary tumors. When they are hyperfunctioning and secrete in excess some of the hormones from the anterior lobe of the pituitary gland, they produce endocrine syndromes. Thus, eosinophilic adenomas will produce acromegaly or gigantism and basophilic adenomas will cause Cushing's syndrome or disease. But both they and non-functioning tumors (chromophobe adenomas) by overflowing the sella turcica and compressing the optic chiasm cause the characteristic bitemporal hemianopia (see II pair) and an enlargement of the sella turcica that is observed in the radiological examination of the skull.

2. Infratentorial posterior fossa tumors. During their course, these tumors generally produce marked intracranial hypertension that appears early. They are the most common in childhood. They can settle in:

a) Cerebellum. They produce a cerebellar syndrome, which will have different characteristics depending on whether the lesion is located in the vermis or in the hemispheres (see "Cerebellar syndrome" in this same chapter).

b) Cerebellarpontine angle. The most frequent and characteristic is the neuroma of the VIII nerve, which is a benign tumor of slow evolution, which is manifested successively by unilateral ringing in the ears (tinnitus) and then nervous deafness on that same side. It can also injure the other cranial nerves that emerge in this region and compress the cerebellar hemisphere on the same side. In this way, the following symptoms can be added to the previous symptoms: peripheral facial paralysis (VII nerve): facial paresthesia with or without abolition of the corneal reflex and sensory disorders (V nerve), and a cerebellar syndrome.

c) Fourth ventricle. They cause an early and severe intracranial hypertension syndrome, generally without other accompanying signs.

Complementary exams

1. Fundus of the eye. We have already highlighted the importance of the fundus in intracranial hypertension when we referred to papilledema as its primary symptom. In the presence of a subject with violent or continuous headaches, we must systematically perform fundus examinations.

2. Lumbar puncture and CSF examination. A lumbar puncture should never be performed on a patient with well-established intracranial hypertension if he or she is not admitted and under the care of a neurosurgical service. Lumbar puncture and CSF extraction can produce an imbalance in intracranial pressures, precipitate wedged bulb, and death of the patient, especially if the suspected cause is a brain tumor. If it is meningoencephalitis or subarachnoid hemorrhage, lumbar puncture is indicated and only requires normal precautions.

3. Electroencephalogram. It may require a slow wave focus at the tumor site. When intracranial hypertension is marked, there may be a generalized slowing of the waves.

4. Simple skull x-ray. It has little value if there is no contrast, so it is often negative.
Calcified hemangiomas can sometimes be observed; the accentuation of digital impressions (bony depressions corresponding to the gyri) in children; the widening of the optic foramen in tumors of the nerve and the optic chiasm.
titic. In tumors of the sella turcica, balloon dilation will be observed in cases of intrasellar tumors and crushing in extrasellar tumors.

5. Carotid angiography. It can be performed when, from a semiological point of view, there is a possibility of locating the tumor above the tentorium cerebellum; in cases of subarachnoid hemorrhage, to detect aneurysms of the circle of Willys. In cases of tumors it may show displacement of vessels or hypervascularized areas, and in cases of abscesses it will show an avascular area. It is not useful in metastases.

6. Pneumoencephalogram. It is a specialized examination that must be performed by a neurologist or neurosurgeon. It can show hydrocephalus due to intracranial hypertension, a deformity of ventricular images in the case of a supratentorial tumor, lack of filling of the fourth ventricle in some stem tumors, etc.

7. Brain scan. Examination that may show an accumulation of radiopharmaceutical in the area of the tumor. It is useful to differentiate primitive tumors from metastatic ones, as well as when there are doubts about whether a lesion may be vascular or tumorous in nature.

8. Computed axial tomography. It allows tumors (neoplasms or abscesses) to be located without risk to the patient.

Etiology

Among the causes of intracranial hypertension we have, in order of frequency:

I. Endocranial tumors. They constitute 80% of the causes of intracranial hypertension; They can be classified as primitive and secondary.
  A. Primitive tumors. They are classified according to their origin into:
    1. Gliomas of the nervous tissue, which can be of different histological types; example: astrocytoma (the most common of them and histologically benign), glioblastomas, oligodendrogliomas, etc.
    2. Meningiomas, tumors of the meninges that are most common at the level of the convexity of the brain.
    3. Nerve tumors; example: acoustic neuronma.
    4. Pituitary tumors; example: eosinophilic and basophilic adenomas.
    5. Vascular tumors.
    6. Sarcomas.
    7. Papillomas (choroid plexuses).
  B. Secondary tumors. It is necessary to look for the primitive in some viscera (such as the lung or kidney in men and the breast in women).
I. Meningoencephalitis.
III. Brain abscesses. In them there is the antecedent te of a neighboring infection (contiguity routes) such as otitis, mastoiditis, sinusitis, or the presence of a hepatic, subphrenic, appendicular or pulmonary abscess (hematogenous route).

Abscesses give localization symptoms the same as tumors but are accompanied by fever and leukocytosis.

IV. Tuberculones. These inflammatory tumors are rare and when suspected it is advisable to look for a history of pulmonary, lymph node or bone tuberculosis.

COMATOSUS SYNDROME

Concept

It is known as a coma (from the Greek kóoma: lethargy, deep sleep) a state of unconsciousness in which the functions of relational life (motility and sensitivity) have been lost, but those of vegetative life are preserved. It is a serious and frequent condition, which can be due to the most varied causes, both intracranial and extracranial.

It is convenient to clarify that the following states that can simulate it are excluded from the concept of a coma:

1. Hysterical crises.
2. Epileptic seizures accompanied by loss of consciousness.
3. Narcolepsy, a disease that causes deep and uncontrollable sleep, so the subject can fall asleep in the most inappropriate situations (eating, standing, etc.), but can be woken up.
4. Syncopal seizures of various causes (orthostatic hypotension, vagal seizures, Stokes-Adams syndrome and other arrhythmias, carotid sinus syndrome), which are usually of short duration.

Syndromogenesis or pathophysiology

1. Consciousness and relationship life. The state of consciousness depends on the activation of the cerebral cortex by a complex system that is located along the brain stem: the ascending reticular activating system. The activation of the cortex depends on the arrival of diverse sensory stimuli through the reticular substance and the multiple collateral pathways that provide sensory pathways to this system. This is the extralemniscal system.

In the thalamus there are nonspecific nuclei, representing tatives of this activating system. In the midline of the thalamus, a system of fibers originates that is diffusely distributed in the cerebral cortex, from which fibers originate that in turn have action on the reticular substance.

In animals, the section of this ascending activating reticular system, without injury to the lemnisci, permanently deprives the cerebral cortex of stimuli and produces constant drowsiness, a state similar to that of coma. In contrast, lesions of the lemniscus cause anesthesia, but not unconsciousness or drowsiness.

Comas depend on the involvement (whether due to direct injury, toxic impregnation, etc.) of the ascending activating reticular system or the projections that continue it, as well as the central vegetative regulation systems also located in the diencephalon-brainstem axis (Barraquer).

2. Vegetative alterations. At the level of the brain stem, there are intricate systems that regulate awakening and relationship life, the ascending reticular activating system (RAAS), and on the other hand, neuronal groups that intervene in the regulatory mechanisms of major vegetative functions, which explains the almost constant association in the course of coma, of consciousness disorders and neurovegetative alterations. The two poles of the brainstem - the upper and lower parts - play a role in the mechanism of these disturbances.

a) The upper pole of the RAAS, with the hypothalamic complex and the paraventricular nuclei, are responsible for thermal elevations and secondary elevations in blood pressure, water disorders, and alterations that appear in the EEG.

b) The lower pole of the RAAS, bulbo-pontuberancial, is responsible for keeping the aerodigestive carreffour in proper functioning (J. and G. Godlewski). This explains the swallowing disorders, largely responsible for respiratory disorders, characterized by pharyngotracheobronchial plugging. Dorsal recumbency favors these disorders, which quickly worsen due to alterations in the rhythm and amplitude of pulmonary ventilation.

The dysregulation of the respiratory, cardiovascular and bulbar vasomotor centers explains the severe and irreversible respiratory disorders, the circulatory collapse and the death of the patient.

Syndromography or positive diagnosis

Different degrees of coma. We believe that there are two previous states before reaching coma, which are obtundation and stupor or waking coma. The latter has been called "waking coma" in certain situations, but we consider that this should not be the case according to the postulates stated in the concept of coma.

Let's see below the different situations that can arise (fig. 54. 10).

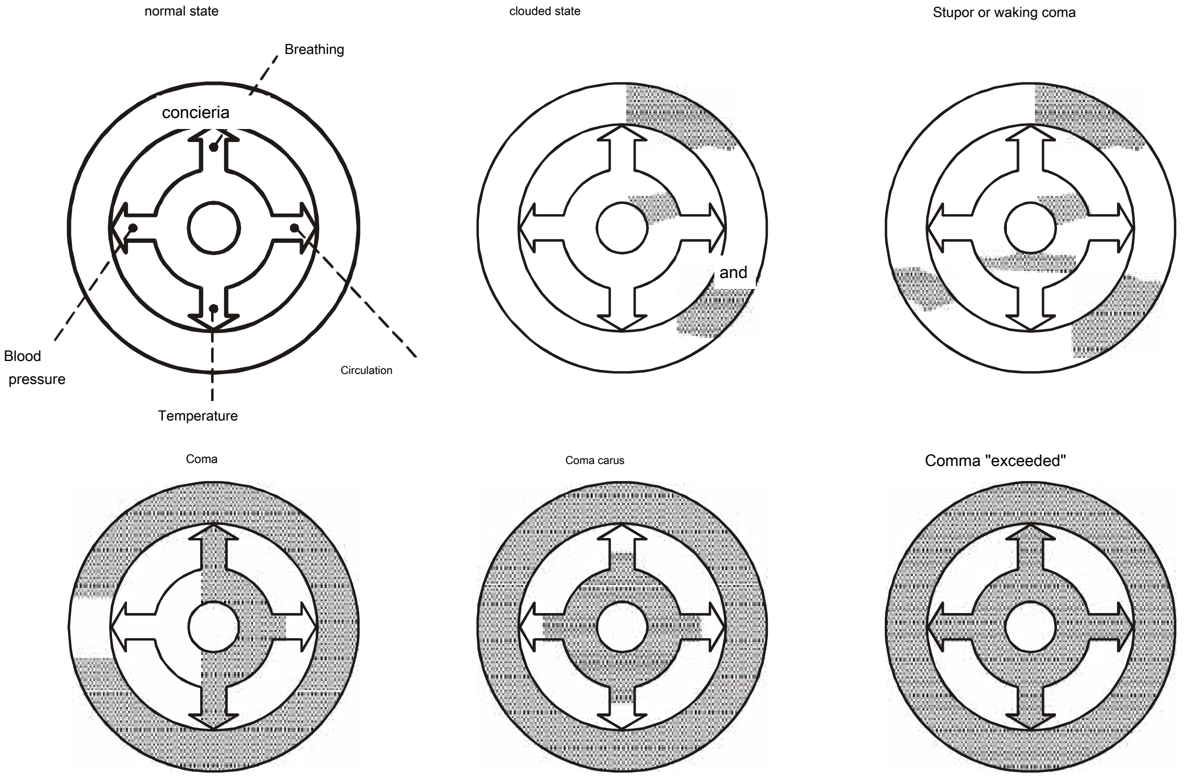


Fig. 54.10 Scheme of the varieties or gradations of the coma state. (Dotted lines indicate the sector(s) taken out of action.)

1.Obnubilation. It constitutes one of the first manifestations of what has been agreed to be called "dissolution of the state of consciousness." The patient lies in bed, mobilizes the eyeballs. "He lazily half-opens his eyelids," but shows no interest in his surroundings. To obtain an answer it is necessary to question him repeatedly, which shows, among other things, that there is a decrease in attention. The answers obtained are brief, hesitant, but often correct.

2. Stupor (some speak of a waking coma), It is manifested in the patient by an increase in sleep (hypersonmnia). However, with certain excitements it is possible to wake it up. His voice tone is low, he can respond to intense stimuli, although such responses are erroneous and interrupted by prolonged and repeated yawns. The patient is not able to follow orders. Sometimes you try to do them but they ARE wrong.

3. Eat. The patient presents a profound depression of consciousness; with the mouth half open, through which some secretions sometimes slip, or, on the contrary, he keeps it closed. His eyes are closed.
When faced with very intense excitement, the patient may respond with a monosyllable or an excitement.
We cannot communicate with the patient. exists loss of motility, sensitivity, but vegetative functions are preserved, although sometimes with alterations.

4. Comna carus. It is a degree higher than the previous one. The patient lies inert, with his mouth half open. It is insensitive to all stimuli. We can't communicate with him.

5. Coma surpassed (depassé). Patient survival is only maintained while resuscitation techniques are applied. Everything is abolished, finished; even vegetative functions.

Coma can be reversible or irreversible. One can only speak of irreversible coma when it is proven that there is brain death, which is manifested by a deep coma, with bilateral paralytic mydriasis, absence of any deep or superficial reflex, absence of all movement and breathing, and a flat electroencephalographic tracing, without any type of brain wave.

This criterion has emerged from advances in intensive therapy and resuscitation that allow breathing, nutrition, hydromineral balance, etc. to be artificially but effectively maintained for some time, despite the existence of brain death. The irreversible coma corresponds to the * exceeded coma of other authors.

Examination of a patient in a coma

As a preliminary step, it requires careful interrogation of family members and friends, in order to specify the conditions under which it occurred:

1. Ingestion of alcoholic beverages.
2. Ingestion of excessive medications due to suicide attempt or not.
3. Head injuries.
4. Previous illness (diabetes, chronic liver disease, nephropathy, high blood pressure).
5. Fever in the previous days.
6. Form of onset (abrupt, slow and progressive, etc.).

The examination must be complete and specify the depth of the coma and the severity of the condition, particularly the vital signs: pulse, blood pressure, respiration.

The neurological examination includes the systematic examination of reflexes, pupils and meningeal signs; the search for signs of motor deficit located in the face, a hemibody or an extremity. Examination of the skull will determine the existence of trauma.

The rest of the examination may show other useful signs to guide us about the cause of the coma: dehydration, Kussmaul respiration, ketonic breath, alcoholic breath, hepatic breath, profuse sweating, etc.

The examination of the fundus of the eye can reveal: papilledema, hemorrhages or exudates, which help to specify the nature of the coma.

The depth of a coma is variable. The extremes are the classic waking coma, whose paradigm is the uremic coma and the carus or deep coma, exemplified by the large cerebral hemorrhage with blood passing to the ventricular system.

Currently, it is about specifying the degree of depth of the coma (in addition to what is explained in the examination of consciousness, in volume 1, Chapter 13) through certain exploratory maneuvers or tests (Jouvet and Dechaume) that allow us to more clearly specify the degree of awareness and the affected levels. Let's look at the most important ones:

1. Tests that explore the degree of involvement of the cerebral cortex (perceptivity):
   - a) Response to a written order.
   - b) Orientation in time and space.
   - c) Simple mental calculation (multiply 5 by 6, etc.).
   - d) Give six names of flowers.
   - e) Execution of a verbal order.
   - f) Reaction to a specific visual form; The patient opens his mouth when a glass full of water is brought to him.
   - g) Search for the reflex of closing the eyelids to the threat.

The normal subject can perform all seven tests; A patient with obtundation is not able to perform the first two tests, but will respond to the others.

A subject in stupor will only respond poorly to the verbal command, and will maintain the response to the visual form and blinking in the face of threat.

A patient in a coma does not respond to any of the tests except in the so-called waking coma, in which he behaves in a similar way to the stuporous patient.

2. Tests that explore the integrity of the brain stem, thalamus and reticular system:
   - a) If the patient is awake, the orientation reaction is used, in which the patient rotates the head and eyes towards the side where a sound stimulus precedes (clap, calling by name).
   - b) If you are not awake, the awakening reaction is used: rotation of the head, opening of the eyes, increase in pulse rate and respiration.

3. Tests that explore the integrity of pain perception, from the spinal cord to the thalamus.

The Pierre-Marie-Foix maneuver or limb clamping is used; The spinal level alone guarantees removal of the limb, but vocal and facial expression responses to pain require integrity of the brainstem and thalamus.

Glascow Coma Scale

It is a standardized assessment tool that facilitates assessments of the level of consciousness in people with current or potential brain dysfunctions.

It is a rapid, objective and reproducible test of the level of consciousness.

Three parameters of the level of consciousness are evaluated: eye opening, best motor response and verbal response. Points are awarded to each category and the total score is determined. The highest possible is 15.

Lower scores are associated with brain dysfunction. Some clinicians use this score to predict patients' prognosis, although this method is not always reliable.

Each category of the Glascow Coma Scale can be scored as shown in Table 54.2.

Table 542 Glascow Coma Scale

Hidden opening	Points	Better motor response	Points	Verbal response	Points
Spontaneous	4	Obeys verbal commands Localized	6	Oriented	5
To verbal stimuli	3	to pain Semi-purposes	5	Confusing conversation	4
to the pain	2	Flexor response	4	inappropriate words	3
No eye opening		Flaccidity	3	Incomprehensible sounds 2	
			1	Tubed	1
				Mutism	

Verbalization is not possible in an intubated patient, even when the patient is fully conscious and alert.

When recording the total score in an intubated patient, "T" is written after the total score to indicate intubation. (Example: 9 T.)

Etiology

Below we present the classification of comas modified by Farreras Valenti:

1. Comas due to diseases of the brain and its coverings:
   - a) Coma due to cerebral vascular accident.
     Hemorrhages: cerebral (apopletic coma), cerebellar, stem, subarachnoid; cerebral thrombosis; transient cerebral ischemia; cerebral embolism.
   - b) Coma in relation to meningoencephalitis.
   - c) Coma caused by tumor (primitive or metastatic).
   - d) Coma due to malignant hemopathies.
   - e) Coma due to status epilepticus.
2. Exogenous toxic comas:
   - a) Coma due to barbiturates and other psychotropic drugs.
   - b) Ethyl coma.
   - c) Morphinic coma.
   - d) Atropinic coma.
   - e) Coma due to organophosphate products.
3. Metabolic comas or endogenous toxins:
   - a) Uremic coma.
   - b) Hepatic coma.
   - c) Coma from hyponatremia and other hydromineral disorders.
4. Endocrine comas:
   - a) Diabetic coma.
   - b) Hypoglycemic coma.
   - c) Myxedema coma.
   - d) Coma due to thyroid storm."
   - e) "Addisonian" comma.
   - f) Sheehan syndrome coma.
5. Comas due to respiratory disorders:
   - a) Asphyctic coma.
   - b) Coma of chronic respiratory failure.
   - c) Coma during the course of the asthmatic state.
6. Commas of physical cause:
   - a) Coma due to head trauma (concussion, subdural hematoma, epidural hematoma).
   - b) Coma due to heatstroke.
   - c) Coma due to heat stroke.

DIGESTIVE SYSTEM. ANATOMY, PHYSIOLOGY, GENERAL PATHOPHYSIOLOGY AND CLINICAL HISTORY

55

ANATOMY AND PHYSIOLOGY

Previous knowledge of the anatomy and physiology of the digestive system is essential both to perform the physical examination and to acquire semiological knowledge. The basics necessary for the physical examination were already covered in volume 1, Chapters 5 and 16, and will also be done in the next chapters of this volume 2, as appropriate, either to clarify the semiogenesis of a particular symptom or, less frequently, due to certain semiographic aspects. In this chapter we will discuss the anatomophysiological knowledge necessary to better understand the general pathophysiology and the performance of some special examinations.

NEOANATOMIC CLASSIFICATION

Knowledge related to Neoanatomy or Physiological Anatomy is very useful in its semiological application, which is why it is convenient to distinguish in parallel, along with the classic segmental classification of the digestive tract described by Testit-Latarjet (fig. 55.1), 1the new anatomical classification based on physiology (fig. 55.2), which divides the digestive tract into the following segments:

- Upper or ingestion segment.
- Middle or digestion segment.
- Lower or excretion segment.

Upper or ingestion segment

Includes the mouth. the oral pharynx and the upper two thirds of the esophagus. It is the ingestion segment of the digestive system; It has a simple function, rather mechanical, and its pathology and semiology are not of major interest in the digestive order itself, except for some specific fundamentally embryological aspects. Its pathology and semiology are common to those of the respiratory system and the mediastinum, as well as to the general pathology of the entire organism.

Middle or digestion segment

It includes the lower third of the esophagus, stomach, duodenum, hepatobiliary system, pancreas, jejunum, ileum, and colon to the

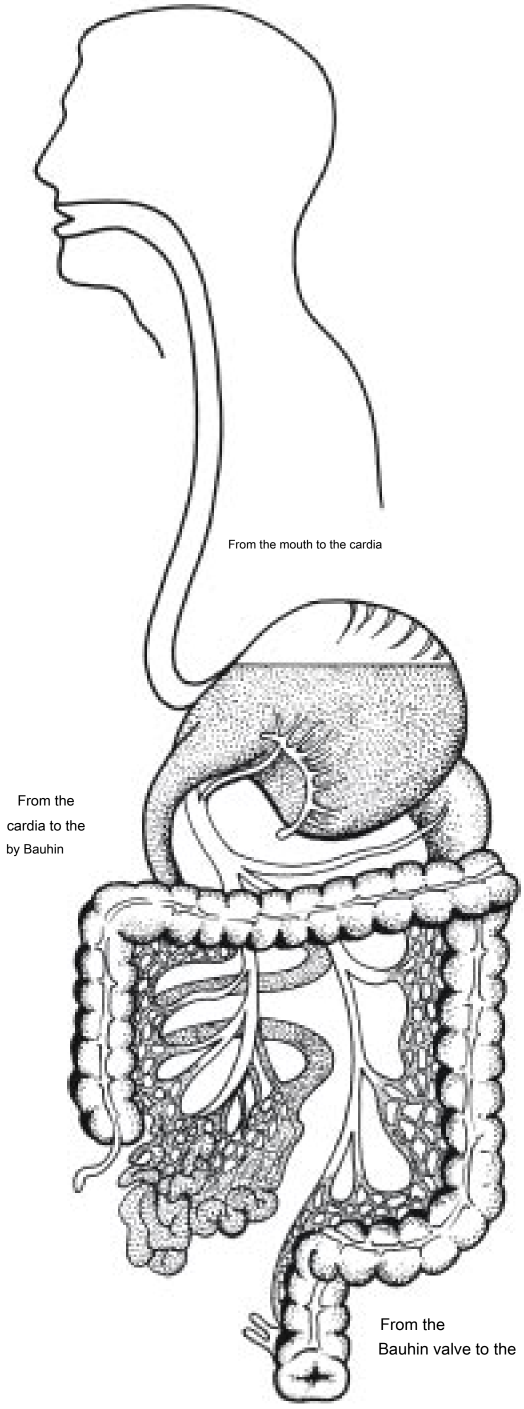

From the mouth to the cardia
From the
cardia to the
by Bauhin
From the
Bauhin valve to the

Fig. 55.1 Classic anatomical segmentation of the digestive tract.

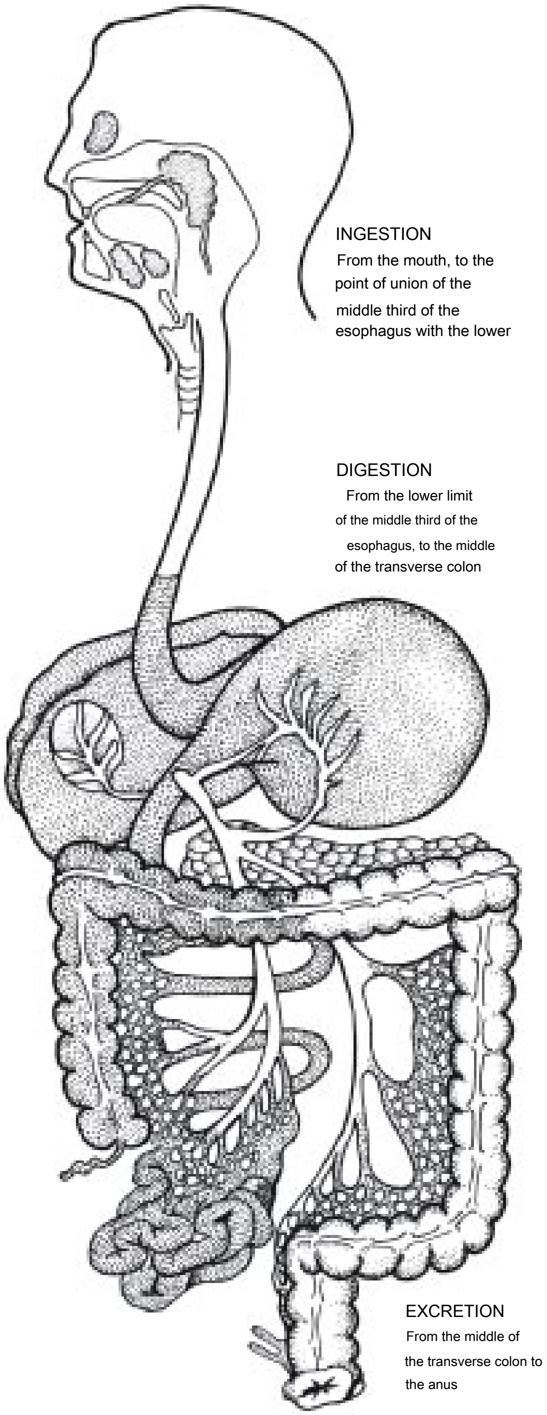

INGESTION
From the mouth, to the
point of union of the
middle third of the
esophagus with the lower
DIGESTION
From the lower limit
of the middle third of the
esophagus, to the middle
of the transverse colon
EXCRETION
From the middle of
the transverse colon to
the anus

Fig. 55.2 Neoanatomical segmentation of the digestive tract.

half of its transverse portion. It is the digestion segment itself (secretion, digestion and absorption); Among its constituent elements there is an evident embryological, anatomical and physiological synergy:

Its pathology and semiology are integrally digestive.

Lower or excretory segment

It includes the distal half of the transverse colon, the descending colon, the sigmoid loop, the rectum, and the anus. It is the excretion segment, whose pathology and semiology, especially in its terminal or anorectal portion, are, in some aspects, common to those of other systems, particularly the urogenital.

This neoanatomical segmentation is closely related to the supposed distribution of the multiple neurogenic nodules that regulate the motility of the digestive tube, and which have been suggested by Keith, parallel to his descriptions of the primitive cardiac tube.

Novoa Santos, as well as Sant' Angelo, describe up to seven segments in the digestive tube, taking into account the distribution of the different nodal points and the corresponding muscular rings; They subdivide the middle segment into four portions, the lower segment into two, and leave the upper segment indivisible. W. Álvarez assures, however, that there has not been a definitive anatomical verification of these nodules.

Although for its study we will group the digestive symptoms following the classic anatomical segmentation: from the mouth to the cardia, from the cardia to the ileocecal valve (Bauhin valve) and from this to the anus, with the aim of making this symptomatic grouping as didactic as possible. It is useful in any case to know the neoanatomical segmentation, since in some cases there is much greater similarity between the symptoms, when they are grouped following the other segmentation. We could cite as an example the common semiogenesis of the so-called juxtacardial symptoms: heartburn, pititus, low dysphagia and gastric regurgitation, which are included in the middle segment of the neoanatomical classification and are, on the contrary, grouped in different portions of the classic segmentation; that is, heartburn, pituitary and low dysphagia, in the first segment, and gastric regurgitation in the second. In any case, we will develop the study of digestive symptoms, following the classic segmental anatomical classification.

GASTRIC ANATOMY AND HISTOLOGY

The stomach from an anatomical point of view has several layers that from the outside in are:

- Mucosa.
- Muscularis mucosae.
- Submucosa.
- Muscular.
- Serous.

Mucous membrane

The mucosa of the stomach is composed of a single superficial layer of epithelial cells, the tunica propria and the submucosa (Fig. 55.3); It begins in the cardia along an irregular or zigzag line (often called the Z" line or “Z-Z" line).

The mucosa is continuous with folds, which flatten considerably when the stomach is distended. In the region of the lesser curvature, where the mucosa is more strongly fixed to the muscular layer, the folds take a longitudinal course, forming what has been called the Magenstrasse.

The folds are generally smallest in the fundus and become larger as they approach the antrum, where they show a tendency to run diagonally across the stomach toward the greater curvature. In addition to these broad folds, the gastric mucosa is further characterized by having numerous shallow invaginations, which divide the mucosal surface into a mosaic of raised areas that vary in shape. When viewed under the magnification of a lens, these gastric areas reveal several delicate projections and depressions, the latter known as gastric pits, crypts, or foveolae. In the depth of these holes, whose thickness and length vary, the glands of the stomach open.

The gastric epithelium, formed by a single layer of columnar cells, begins at the gastroesophageal junction (cardial orifice) with a precise delimitation of the thicker, stratified esophageal mucosa. The epithelial cells are of the mucoid type, containing mucigen granules in their outermost portions and a single ovoid nucleus at their base.

The stomach glands are tubular and can be differentiated into three classes:

1. The cardiac glands. They are limited to a narrow area, 0.5-4 cm wide, around the cardial orifice, they adopt the shape of a spiral and are covered by cells that produce mucus.
2. The gastric or fundic glands (glandulae gastricae propioe). They are located in the fundus and most of the body of the stomach. They are simple branched tubules, quite straight, with a narrow lumen, which reaches downwards almost to the muscularis mucosae. They are covered by three types of cells:
   a) Mucoid cells present in the neck and differing from surface epithelial cells in that their mucigen granules have slightly different staining qualities and their nuclei tend to be flattened or concave (rather than oval) at the base of the cells.

   b) Cells of the second type, principal cells or zymogenous, they line the lower half of the glandular tubules, have a spherical nucleus and contain granules that are highly refractory to light and a Golgi apparatus whose size and shape vary with the state of secretory activity. They are believed to produce pepsinogen (precursor of pepsin).

   c) The cells of the third type, the parietal cells, are larger and generally located far from the lumen to which they are connected by extracellular capillaries, which arise from intracellular canaliculi. Its intraplasmic granules are strongly eosinophilic and less refractory to light than those of cells.

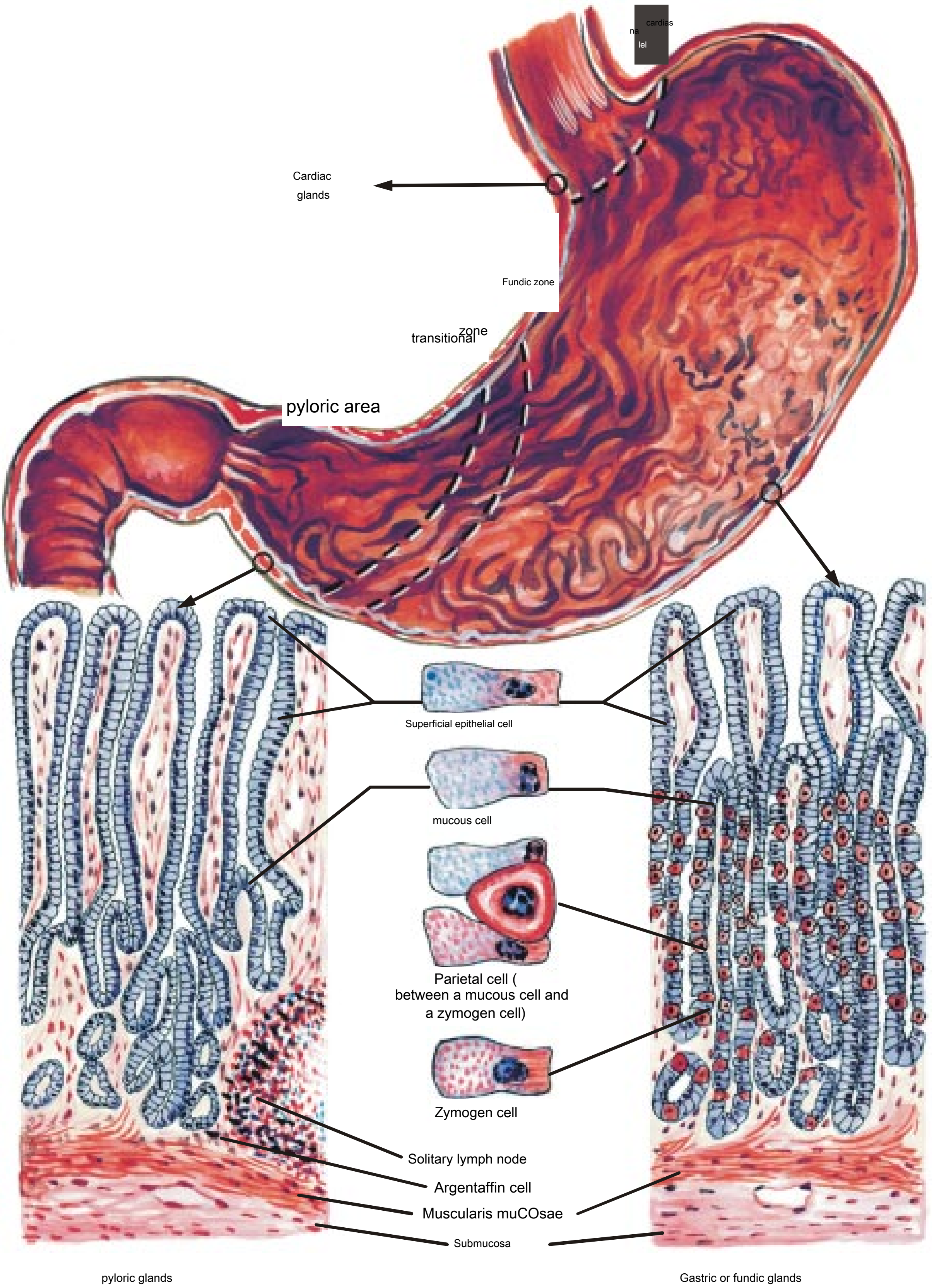

cardias
na
lel
Cardiac
glands
Fundic zone
transitional zone
pyloric area
Superficial epithelial cell
mucous cell
Parietal cell (
between a mucous cell and
a zymogen cell)
Zymogen cell
Solitary lymph node
Argentaffin cell
Muscularis muCOsae
Submucosa
pyloric glands
Gastric or fundic glands

Fig. 55.3 Stomach mucosa.

main. Histochemical studies have shown that parietal cells produce hydrochloric acid.

3. The pyloric glands. They are located in the pyloric region, but also spread towards a transition zone in which glands, both gastric and pyloric, are found, and which extends diagonally and distally from the lesser to the greater curvature. The tubes of the pyloric glands are shorter, more tortuous and less abundant, and their ends are more branched than in the case of the fundic glands. These glands are lined by a single type of cell, which resembles, and may actually be identical to, the mucous cells in the neck of the fundic glands.

Another type of cell, the argentaffin cell (argentophila), lies scattered on the basement membrane of the pyloric glands, but can sometimes also be found in the gastric glands. It has been shown that they produce gastrin, a gastric hormone that stimulates the formation of hydrochloric acid.

Muscularis mucosae

The muscularis mucosae is composed of a thin layer of smooth muscle fibers that separates the mucosa from the submucosa.

Submucosa

It is composed of connective and elastic fibers that allow the mucosa to follow the movements of the muscles under the influence of tone and contractility.

It includes the vessels and lymphatics, as well as the Meissner nerve plexuses.

Muscular

It is made up of smooth muscle fibers arranged in three layers:

1. Circular (median), which covers the entire stomach and is particularly reinforced in the vicinity of the pylorus, where its fibers form the sphincter.
2. Longitudinal (superficial), which covers a part of the stomach, is continuous with the longitudinal fibers of the esophagus and is divided in the cardia into two bands: a denser one, which follows the lesser curvature and another thinner one, which crosses the greater curvature and reaches the pylorus. Therefore, the median areas of the anterior and posterior surfaces do not present longitudinal fibers. The two bands meet in the pyloro and contribute to form the pyloric sphincter through a few fibers and are continuous with the longitudinal fibers of the duodenum.

3. Oblique (deep), which is continuous with the circular muscles of the esophagus. It is thicker in the region of the fundus, where the fibers are arranged in a fan on the anterior and posterior surfaces, disappearing towards the pylorus. There are no oblique fibers on the lesser curvature. The region of the pyloric sphincter is not continuous with the circular muscles of the duodenum.

Serous

It covers the entire stomach, being directly applied, except on the curvatures, where it forms: on the lesser curvature, the lesser omentum, and on the greater curvature, the greater omentum.

Gastric vascularization and innervation are carried out from the seromuscular layer (which is where the thick vessels and nerves arrive) towards the mucosa, starting from the first very fine branches until reaching the mucosa. These branches are formed by fibers: sympathetic (splanchnic nerves) and parasympathetic (pneumogastric nerves).

These nervous fibers, sympathetic (adrenergic) and vagal or parasympathetic (cholinergic), join together in the thickness of the stomach wall forming a wide network that is accepted to be grouped into three plexuses:
subserous (Swabach), myenteric (Auerbach) and submucosal (Meissner).

Splanchnic, sympathetic or adrenergic fibers act as inhibitors, while parasympathetic, vagal or cholinergic fibers act as excitators.

GASTRIC PHYSIOLOGY

Gastric functions are primarily motor and secretory. Their purpose is to allow the organ to receive the different categories of food and to physically and chemically transform these foods for their introduction into the small intestine, where they will be completely digested and absorbed, and their waste will pass to the colon to be evacuated. The absorptive functions of the stomach, which are only exerted for water, alcohol and certain sugars, are insignificant and of no practical importance.

These functions also ensure protection against certain bacteria through gastric acidity. If the possibilities of receptibility and gastric digestion are exceeded, the ingested substances are rejected through vomiting (gastric indigestion).

It is therefore advisable to study:

-Gastric motility, its mechanism and its disturbances

Cions.

- Gastric secretion, its mechanism, the processed product, that is, gastric juice, the means of its study and the consequences of secretory disturbances.

Motor skills

The motor role of the stomach consists of:

1. The reception and momentary conservation of food: reservoir function.
2. The modification of foods to facilitate their entry into the duodenum: mixing function.
3. The evacuation of the prepared chyme: function of va- foundation.

reservoir function

The reservoir function develops mainly in the fundus and in the upper part of the stomach, in which the muscles are thinner and the contractions are less vigorous than in the other regions. A certain relaxation allows an increase in gastric capacity during food ingestion, which prevents an excessive increase in gastric pressure. Relaxing the abdominal wall simultaneously prevents an increase in abdominal pressure.

Mix function

The mixing function takes place in the lower half of the stomach and especially in the antrum. The food that reaches the stomach thus tends to occupy the central part of the food mass, which comes from the lowest part. The liquids float temporarily in the upper part, but are progressively mixed with the mass until the chyme reaches a liquid or semi-liquid consistency that allows its entry into the antral region, where a pressure gradient system makes it possible to travel until it is evacuated into the duodenum (emptying function).

These functions are presided over by two types of motor activity: tone and peristalsis.

The tone represents the state of permanent intrinsic tension: the rhythmic contraction occurs according to a state of optimal tension (Cannon). Four types of stomach can be recognized through radiological study: hypertonic, orthotonic hypotonic and atonic (Fig. 55.4).

Peristalsis is produced by contractions that surpass the pylorus, particularly active at the antrum level. It occurs in a fasting state and after ingestion of liquids and solids. Several types of peristaltic contractions or waves are distinguished: type I, 20 s, which are used for mixing; type II, very propulsive, similar to the contractions of hunger; those of type III, slower and overlapping (Carlson).

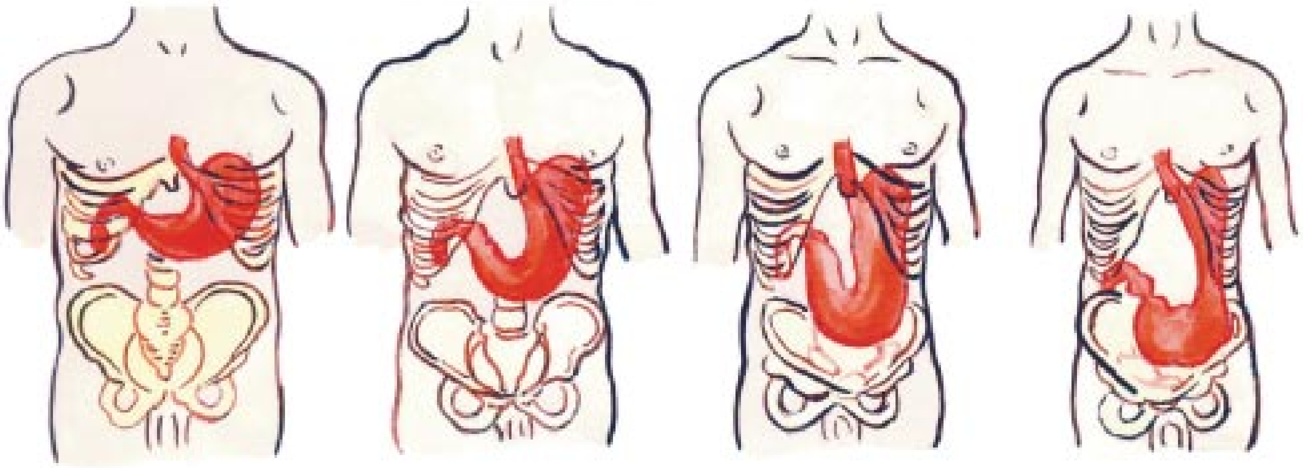
The cause of the motor activity of the stomach is not well understood. It appears autonomous, as it persists after complete denervation. A denervated and transplanted fundic sac contracts simultaneously with the main stomach, suggesting a humoral mechanism.

When food reaches the stomach, tonic and peristaltic motor activity abruptly ceases, allowing an increase of 3-6 cm of water in intragastric pressure.
Then contractions appear, especially pyloric; After 5-10 min, the contractions gain in height and become increasingly deeper and more vigorous when they reach their maximum, two or three large waves are followed 10 s apart by numerous smaller contractions.

Empty function

Gastric emptying occurs through a pump mechanism with a periodic increase in intraluminal pressure above duodenal pressure.

Fig. 55.4 Types of stomach.



Hypertonic stomach

Orthotonic stomach

Hypotonic stomach

Atonic stomach

There appears to be a succession of high and moderate pressures and a mechanism of pressure gradients between the duodenum and the pyloric sphincter. Hunt has established an exponential mathematical relationship in relation to gastric emptying. This starts quickly and then continues more and more slowly. It can be carried out due to a large number of physiological and pathological circumstances. Among the first, the role of intestinal receptors is certain, which are stimulated by distention, fats, osmotic pressure of solutions and perhaps by enterogastrone (Ivy). Varay has personally studied the role played by bile salts, whose intravenous injection produces rapid gastric evacuation, usable in radiology. Among the pathological factors, lesions of the pyloric region and its vicinity are the most frequent cause, as well as certain humoral disorders (hypokalemia, diabetic acidosis).

Among medications, cholinergics increase motor activity and anticholinergics and atropine decrease it. Vagotomy reduces motor activity and makes gastric evacuation difficult.

Pathophysiological consequences

The complexity of the mechanisms that govern gastric motility allows us to understand the diversity and number of causes that can disturb it.

All organic lesions, especially those located in the most active regions (antrum and pylorus) can influence the innervation, modifying the movements and evacuation of the stomach.

It seems that the relationship between the various types of basal behavior of gastric motility and the classification of stomachs into hypertonic, orthotonic, atonic, hypotonic (Schlesinger) can be preserved. But every motor abnormality must be explored to look for organicity, and if it is not discovered according to physiological reasoning, numerous motor or sensorimotor dyskinesias (dyskinetic syndromes) may be related to gastritis, unknown ulcers and humoral or general disturbances. That is, gastric motor functions are influenced by numerous factors, particularly emotional and psychological. The works of Wolf and Wolf have demonstrated in a singular patient the role of psychoemotive aggressions on motor activity and stomach evacuation.

gastric secretion

Beaumont (1833): Pavlov (1910): Heidenhaim; Le Barre, Edkins (1900-1910); and later, Ivy, Dragstedt, Komarov, Lambling, are some of the authors who have made the most contributions.

Pavlov's works have demonstrated the secretory response to psychic incitements, the role of the vagal in the transmission of excitosecretory influences, the secretory response to the presence of food in contact with the antral mucosa and secretory inhibition due to the presence of hydrochloric acid in the stomach.

In the mechanism of gastric secretion there are three phases: cephalic (vagal action); gastric and intestinal (according to Dragstedt: cephalic, 45%; intestinal 10%).

The cephalic phase, also called initial, psychic or vagal, reaches the first half hour or three quarters of an hour of digestion. It is verified by psychic, visual or olfactory evocation. Pretend meals in animals that have undergone an esophagostomy demonstrate the role of oral tactile or gustatory sensations and that of chewing. Section of the vagus in experimental animals suppresses this type of cephalic or psychic secretion. The reflex action is not only born in the mouth, but it can be born in the cortex (example: thinking about food), or in the sense organs (example: seeing an appetizing dish, or feeling the smell of food or sweets, etc.), therefore, the influence of the brain centers is undeniable. The appearance of acute ulcers in conditions of marked stress (systemic or psychological, such as emotional tension, anger, upsets, conflictive situations, etc.) argues in favor of the influence of the cortical and subcortical brain centers in this phase.

Insulin hypoglycemia (Le Barre) with blood glucose below 50 mg% causes acidic gastric juice of purely nervous origin (Hollander test).

The gastric, medium, chemical or hormonal phase begins approximately half an hour after eating the test breakfast and lasts up to two and a half hours. His character is growing; reaches the maximum at 60-90 min. This secretion is established by the contact of the food with the gastric mucosa, at the expense of different factors that seem to influence:

1. Direct (mechanical) excitation of the glands by food.
2. Absorption by the blood and conduction to the glands of secretagogue substances present in food.
3. Stimulation of the afferent nerves (central reflex arc).
4. Local nervous stimulus, without going to the centers (local reflex arc).
5. Release of a hormone, coming from certain parts of the gastric mucosa of the stomach, which would act on the glands.

Of all these factors, the hormonal type seems to be the most important, and thus it is pointed out that gastrin or gastric hormone (discovered by Edkins) is formed in the pyloric antrum by the contact of food with the gastric mucosa, passing said gastrin into the blood, and

Through the blood, it acts on the chlorhydropeptic glands of the body and fundus; This is proven when the antrum is removed from experimental animals, and in the clinic it is observed when performing partial gastric resection or subtotal antral gastrectomy, for the treatment of gastroduodenal ulcer. In both cases, the absence or marked decrease in secretion is verified.

Inhibitory factors act in another sense: the role of hydrochloric acid as such (role of antral pH) has been proven. The problem of an inhibitory hormone must be taken into account (Harrison, Duval and Price, Thompson), whether mobilized by acidic pH, whether it exists in the gastric juice (Branschwig).

The intestinal or terminal phase begins after two hours and lasts for half to three quarters of an hour. The secretion could be due to a secretagogue action of food origin and transmitted through the blood or produced by a hormonal mechanism, analogous to that of the gastric phase, under the stimulus of a substance equal to or similar to gastrin, which would be generated in the duodenum when it receives the gastric contents. Next to the excitation factor there is another inhibition factor; The presence of fats and sugars in the duodenum produce the so-called enterogastrone (Ivy), which inhibits gastric secretion. The existence of urogastrone is also noted, which also inhibits gastric secretion.

Gastrointestinal hormones will be discussed in more detail in Chapter 63, dedicated to the main syndromes of the digestive system.

Another interpretation is derived from works by Dragstedt: The cephalic and gastric secretory phases are not independent, but are in close collaboration and interdependence.

Finally, the action of histamine present in the gastric juice (not demonstrable in the blood during digestion, which may be due to the lack of sensitivity of the methods, since there is excretion in the urine, proven by Ivy and Schayer with marked histamine), has not been established, nor has the action of acetylcholine. It has been argued that gastrin could release histamine. For Babkin, histamine would be, ultimately, the direct excitosecretory agent of parietal cells, common to all types of stimulation.

The discovery of the clinical Zollinger-Ellison syndrome has highlighted the problem of stimulation by a pancreatic intrinsic factor, different from insulin secretion.

gastric juice

Gastric juice collected by tube is a mixture of gastric secretion, saliva, and duodenal regurgitation. Obtained from the stomach or isolated pouches, it appears transparent, colorless, occasionally altered by suspended mucus or sloughing epithelial cells.

Its density is 1,002-1,007, the freezing point is very similar to that of plasma and its viscosity and adhesiveness are due to mucus. The volume collected per 24 h in the normal fasting individual is 1-1.5 L, increasing to 2.5 L and more due to stimulation with food.

It is made in two totally different areas: the antrum and the fundus, which contain specific glands.

The anthropyloric glands, made up of small straight tubes, contain mucous cells on the surface and lighter cells or mucoid cells in the depths. The basal cells of the mucosa secrete gastrin.

The fundic glands constitute a superficial layer, whose mucoid cells secrete a thick mucus through their mucosal epithelium and a soluble mucus through their neck, as well as sodium and potassium bicarbonate at a constant concentration: the whole constitutes the primary alkaline secretion. Below there is a branched tube lined with granular cells: the main cells, which secrete pepsin (inactive pepsinogen grains, which transform into pepsin upon contact with HCl after vagal excitation) and the parietal delomorphic clear cells, which contain fine granulations and secrete HCl.

The formation of HCl is very complex. The H ion comes from water and its processing produces an OH residue that is probably buffered by a carbonic anhydrase. The Cl ion comes from NaCl and the Na* residue intervenes in the buffering of the OH ion to form NaHCO, while HCl comes out of one pole of the gastric cell, and NaHCO comes out of the other pole; These processes are accompanied by a phenomenon of polarization of the cell membrane, since polarity in the opposite direction suppresses acid secretion. This has an isotonic concentration with the plasma.

The composition of gastric juice, contrary to what is believed, somewhat superficially, is not limited to HCl and pepsin.

Hydrochloric acid, whose maximum concentration reaches 143 mEq/L in humans (Lambling and Bernier), exists in free form and combined with proteins (mucin hydrochloride). But there is also a primary alkaline secretion, which is a mixture of sodium bicarbonate (20 mEq/L) and mucin (alkaline mucins, 20 mEq/L and 90 mEq/L). To explain the variations in the acidity of the gastric juice, Roseman's theory of variations in the composition of the product is no longer accepted, since the secretion of Cl is constant and that of H variable.

On the contrary, the theory generally accepted by Pavlov-Hollander is based on the fact that the secretion of HCl by the parietal cells is at a constant concentration: 166 mEq/L in the dog (that is, Ci: 166 mEq/L, H: 159 mEq/L and K: 7 mEq/L) (Pavlov-Hollander theory) and 143 mEq/L in man (fig. 55.5).

According to this isochlorhydria theorem, by Pavlov-Hollander, the isotonic primary acid secretion of the plasma at constant concentration is mixed with the alkaline primary secretion, a mixture that tends to a partial neutralization of the excessive secretion (Lambling). In the most common case, the amount of acid ions is greater than that of alkaline ions. In certain circumstances, acid secretion is completely neutralized by alkaline secretion.

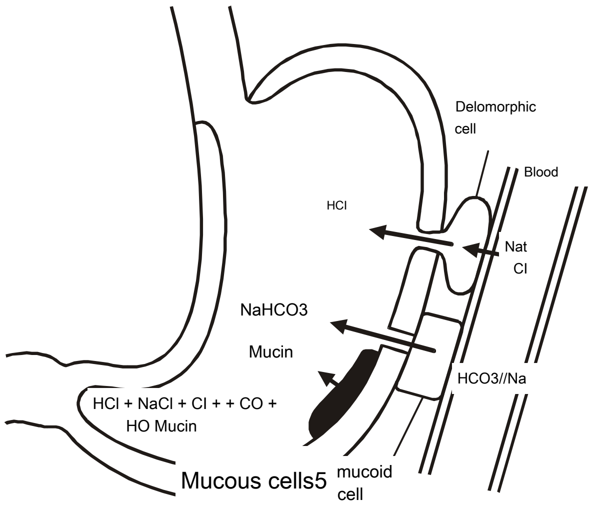
Other theories explain the variations in the electrolyte composition of gastric juice in a different way. Teorelt's theory is based on the possibilities of diffusion of ions through the mucosa: the H ions secreted by a pure primary secretion of HCl of variable concentration are capable of diffusing out of the mucosa and being replaced by Nat ions. Hirchowitz's "gastron" theory, which considers a functional unit whose primary secretion would not be acidic and sodium would be exchangeable with H ions during the passage through the tube, has only limited acceptance.

HCI secretion is an intermittent phenomenon and there are substances that stimulate it to different degrees;
Thus, water offers minimal stimulation while parenteral histamine offers maximum stimulation.

The HCI's functions are:

1. Lower the pH for optimal pepsin action.
2. Activate pepsinogen.

Fig. 55.5 Schematic representation of the Pavlov-Hollander theory.



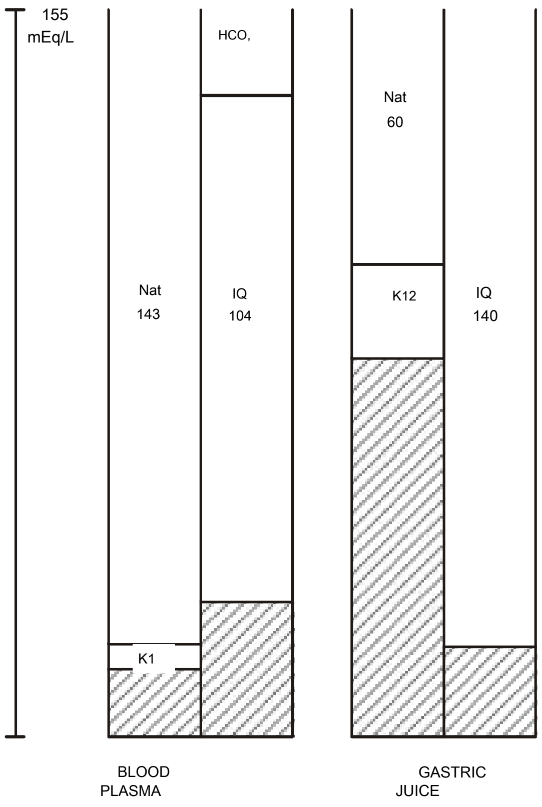
3. Transform proteins into acidic metaproteins.
4. Sterilize the gastric contents.

Gastric juice also contains total chlorides, phosphates and sulfates in small quantities. The cations are sodium (very variable, 10-12 mEq/L), magnesium and calcium, at a concentration equal to or lower than the body's fluids. Potassium alone has a concentration two or three times higher than that of the blood: any loss of gastric juice results in severe potassium depletion (Fig. 55.6).

Gastric juice also contains ammonia. This is formed by urease, present in large quantities in the cells of the surface epithelium. In humans, 10% of urea is metabolized in one hour by gastric urease. Elevation of blood urea increases gastric ammonia production and lowers acidity.

The mucus is secreted in the following way: the mucous cells on the surface produce a substance that, attacked by proteolytic enzymes, is transformed into a mucoprotease; The other precipitated part is the apparent mucus. Mucoid cells in the neck of the fundic glands produce mucoprotein. The set - mucoprotease, apparent mucus and mucoprotein - constitutes gastric mucin (fig. 55. 7).

Fig. 55.6 Comparison between the electrolyte concentrations of plasma and gastric juice.



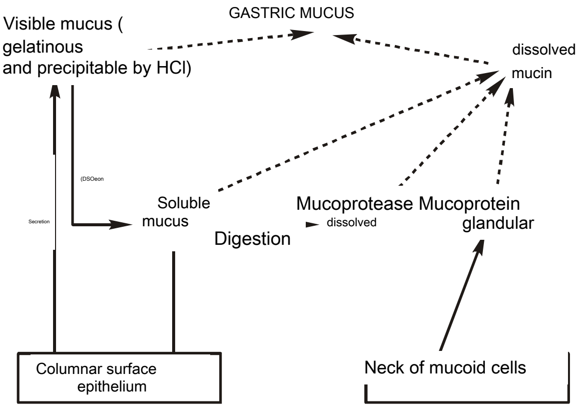


Fig. 55.7 Glass's diagram of the formation of gastric muCus.

Histochemical characteristics differ depending on the muciparous cells. In the fundus, mucoid cells do not contain polysaccharides of acidic groups, while goblet cells do. In the antrum, the mucus of the crypt and surface cells is formed by glycoproteins with masked sulfide groups. The bottom of the glands contains sulfated polysaccharides (A. Gerard).

The enzymes are mainly pepsin, secreted in inactive form, and pepsinogen, which during the resting period accumulates near the apical membrane of the main cell in granules, in the form of a deposit.

Pepsinogen is transformed into pepsin by the influence of HCl. Pepsin only appears after the sixth month; The infant has a weak proteolytic diastase: cathepsin, responsible for the neutrality of its gastric juice. Pepsin is found in small quantities in the blood and in greater proportion in the urine (uropepsin).

The lab ferment existing in the stomach of ruminants transforms the milk into a solid mass that allows the whey to overflow. It is tended to be considered that the lab ferment is identical to pepsin in children and that it is only found in the stomach of some ruminants: cows and lambs.

A protein anabolic factor, intrinsic antianemic factor, is also found in gastric juice.
It interacts with vitamin B, preparing it for absorption.
Sorption in the intestine.

Other proteolytic enzymes exist in gastric juice:
cathepsin, a gelatine, and non-proteolytic enzymes;
a carbonic anhydrase, the urease already mentioned, sometimes lysozyme and a lipase of duodenal origin.

Summarizing what was said above, gastric digestion is carried out through two functions: mechanical and chemical. It seems that gastric digestion is a more mechanical than chemical phenomenon, which involves food to a state of maximum grinding that facilitates its subsequent chemical digestion in the duodenum, a fact proven experimentally by continuing gastric digestion in the stomach without acids and poor in ferments.

Likewise, the digestive adaptation of the extensive partial gastrectomy is verified in the clinic. On the other hand, gastric pathology is more motor than secretory or chemical, that is, there is more gastric dyskinesia than dyspepsia per se. In any case, both functions complement each other to carry out normal digestion and thus the stomach fulfills its task of mixing, diluting and breaking down the food before it leaves it towards the upper parts of the small intestine.

ANATOMY AND PHYSIOLOGY OF THE BLADDER AND BILE TRACT

Anatomy

The vesicle is pyriform: it is located on the lower surface of the right lobe of the liver, where it has its bed.

It distinguishes the fundus, the body, the infundibulum or basin and the neck, which continues with its excretory or cystic duct. The capacity of the vesicle is 40-60 mL. It has a fibromuscular layer, a mucosa lined with long columnar epithelium, and a serous layer.

The cystic in its first portion shows a series of spiral valves called Heister's and then a smooth portion, which when joined with the common hepatic, forms with it the common bile duct. In the cystic duct the bile pressure is regulated to about 25 mm.

The liver, about 3 cm long, has at its end a sphincter called Mirizzi's sphincter. The common bile duct is about 6 cm long by 8 mm wide and in relation to the pancreas it has a portion above and another behind it, ending in the second portion of the duodenum, in the ampulla of Vater, where also, with a frequency of 60%, the pancreatic duct of Wirsung empties. The mouth of the common bile duct has a sphincter called the sphincter of Oddi, which plays a very important role in the foundation of biliary drainage.

Physiology

The vesicle has the following functions:

1. Storage and concentration of bile. Bile is secreted by the liver cell, in an amount of 1-1.5 L in 24 hours; This phenomenon is carried out continuously, with bile circulating through the canaliculi and hepatocholedochal duct, but since in the fasting phase the sphincter of Oddi, at the level of the duodenum, is closed, a pressure of about 50-70 mm of water is generated, which forces the cystic duct and the bile is forced to penetrate into the

vesicle, which stores it, while concentrating it in a proportion 8-10 times greater, thus converting it into vesicular bile. This function is carried out by reabsorption of water and some salts such as chloride and sodium bicarbonate, therefore concentrating bile salts, bilirubin, calcium and, to a lesser extent, cholesterol. Approximately 600-700 mL of hepatic bile secreted are reduced to 60 or 70 mL of vesicular bile. The vesicle secretes a certain amount of mucin.

2. Regulation of bile pressure. The pressure at which bile is secreted by the liver cell increases progressively up to about 250-300 mm of water, but remains leveled at the expense of the regulation made by the vesicle with storage and concentration.
   Ligation of the common bile duct in dogs demonstrates that the elevation of bile pressure is controlled by the gallbladder and cystic duct, since said ligation can be tolerated for 24-36 hours without jaundice appearing, despite continued hepatic secretion of bile. The sphincter of Oddi can withstand a pressure of up to 200 mm of water.

3. Acidification of bile. Hepatic bile is alkaline, and its pH decreases in the gallbladder, becoming slightly acidic, thus avoiding precipitation phenomena.
4. Evacuation of bile. The function of the vesicle is to empty its contents into the duodenum during the period of food digestion thanks to stimuli generated therein. The gallbladder evacuation phenomenon is active, due to intrinsic contracture of this organ, and reaches a pressure of 300 mm of water. To explain vesicular emptying, different mechanisms have been pointed out:

   a) Mechanical action. At the level of the duodenum, aspiration, vacuum siphon (perhaps aided by peristaltic shaking movements from the intestine).

   b) Resorption action at the level of the gallbladder mucosa:

   c) Stimulating action of food. Especially fats, which upon contact with the duodenal mucosa produce a hormonal type substance, which when passed into the blood determines the relaxation of the sphincter of Oddi, with the subsequent contraction of the gallbladder. This substance is Ivy's cholecystokinin, which if experimentally injected intravenously, produces biliary contraction.

   d) Action of other substances instilled in the duodenum. Such as magnesium sulfate at 25 6 33%, peptone at 10%, which are capable of provoking the contralateral reflex, consisting of the relaxation of the sphincter of Oddi and contraction of the gallbladder.

   e) Nervous excitement, both of the vagus and the sympathetic. It can cause slight contractions of the gallbladder temporarily, although contracture appears to predominate due to vagal action and relaxation due to sympathetic action.

In relation to the bile ducts, it is estimated that the sphincter of Oddi contracts by vagal action and relaxes by sympathetic action. Likewise, drugs such as bantine, atropine, aminophylline, amyl nitrite, trinitrine, etc., relax the sphincter of Oddi, while other drugs such as paregoric elixir, morphine, prostigmine, meperidine, pilocarpine, etc., do so.

they bring

PANCREATIC FUNCTION

The physiological knowledge that there are multiple hormonal, parasympathomimetic, etc. substances capable of stimulating pancreatic secretion has made their study possible, as well as the determination of their enzymatic values in duodenal juice, blood and urine.

At present, these biological tests constitute the means of exploration that most point towards the existence of pancreatic insufficiency, having somewhat lost their value in studying feces and digestive tolerance tests.

These tests, together with pancreatic scintigraphy, are the ideal means of exploring this organ, which, until now, is difficult to investigate.

It is useful that we know the different stimuli of pancreatic secretion, before learning how to handle the different exploration tests of this organ.

External pancreatic secretion is stimulated by:

1. The vagus nerve, which produces a juice rich in enzymes, by constriction of the pancreatic ducts, causing zymogen granules to come out of the acini except those located near the islets. The vagus also stimulates the secretion of mucus by the mucous cells of the pancreatic ducts.

2. The acidic pH of the chyme, which upon reaching the small intestine causes the secretion of secretin and pancreocymin.
3. Duodenal secretin, which produces abundant pancreatic juice, rich in bicarbonate (hydrolatic secretion).

4. Duodenal pancreocymin, which stimulates the secretion of pancreatic juice rich in enzymes (ecbolic secretion).

5. Glycaemia that stimulates the secretion of pancreatic enzymes.

The most used biological (enzymatic) tests can be classified into three groups:

1. Those that stimulate the volume of pancreatic juice through the administration of secretin.

2. Those that stimulate the production of enzymes (mecolyl, protigmine, pancreocymin).
3. Those that block excretion at the level of the pancreatic duct, such as morphine.

GENERAL PATHOPHYSIOLOGY

In this section we will overall review the functional or pathophysiological alterations most frequently observed in digestive pathology. On this occasion we begin to know the lexicon of functional digestive diseases, that is, those determined by a pathophysiological substrate. These diseases are generally classified as secondary digestive diseases, since the rule is that they are dependent on injuries or functional alterations of other systems.

We will point out, however, the possible existence of functional diseases of a primitive type; that is, apparently initiated in the digestive system itself. Of course, every digestive organic lesion generates pathophysiological alterations in the affected organ; Digestive functional alterations are also observed in a certain segment, which are secondary through reflex or humoral means to lesions or functional alterations of other segments or organs of the digestive system itself.
The fundamental and most frequent pathophysiological alterations are:

- Motor alterations.
- Secretory and absorption alterations.
- Sensitive alterations.
- Mixed alterations.
- Alterations in chewing, swallowing and salivary secretion.

MOTOR ALTERATIONS

They include alterations in the tone of the digestive muscles, which are expressed by hypertonia or hypotonia; alterations in peristalsis, either due to hyperperistalsis or hypoperistalsis, as well as reversive peristalsis, so studied by Álvarez, and, finally, alterations in motility or evacuation, expressed by hypermotility or hypomotility.

Generally, there is parallelism between these alterations, but it may not exist, and different combinations of them may be observed. Altogether, these motor alterations constitute the so-called digestive dyskinesias, a fundamental term that will be used frequently in the course of these studies of digestive semiology, since we must accept the mechanism of motor alteration as an essential and preferential mechanism of digestive alterations.

The intimate mechanism of all dyskinesia, in general, depends fundamentally on the loss of vagosympathetic balance, which is what regulates motor function. At the same time, we must also consider as pathophysiological factors the alterations of the muscle layers, the nervous plexuses, the sphincters and other neuromyogenic elements of the digestive system, as well as the humoral, hormonal and circulatory metabolic alterations. Let us point out, especially because of its extreme importance, the influence of the psychic factor, with the participation of the cerebral cortex and its relations with the midbrain.

As examples of dyskinesias we can mention: gastric dyskinesia secondary to gynecopathy (metroadnexitis, for example), with a predominantly reflex mechanism; intestinal dyskinesia with diarrhea of hyperthyroidism, of reflex and hormonal mechanism in turn; gastric dyskinesia, apparently essential or primary, equivalent to what is called nervous dyspepsia by European schools and in which hypotonia, hypoperistalsis and hypomotility are generally observed; gastric dyskinesias of chronic duodenal ulcer and cholecystitis, in which cases hyperperistalsis with hypermotility or hypomotility is generally observed, depending on the stage of development; Finally, let us also point out the existence of hypertonic or hypotonic vesicular dyskinesias and colonic dyskinesias, among others, irritable colon itself, motor diarrhea and colic paresis.

Sometimes, the motor alteration is discrete and imprecise in its location and characteristics, and is called motor neurosis by the German school.

SECRETORY AND ABSORPTION ALTERATIONS

Hyperfunctional or hypofunctional alterations may be observed, which depend on the greater or lesser functional capacity of the specialized epithelium: secretory or absorptive. In turn, these are grouped, according to their location, into gastric, duodenobiliopancreatic and intestinal. These secretory alterations are noticeable, globally or partially, in relation to the different ferments or enzymes of each gland, with or without parallelism between them; They can be considered together, like true dyspepsias and dystripsias, since the functional alteration consists of the deviation or dysfunction of the ferments or enzymes. In the stomach, for example, hypersecretion with hypochlorhydria and vice versa can be observed.

As intimate mechanisms of these disorders, vagosympathetic alterations, histological alterations of the glands themselves, humoral, hormonal, metabolic and circulatory alterations, as well as psychological alterations, should also be noted.

As examples of secretory alterations due to hyperfunction, let us cite gastric hypersecretion with hyperchlorhydria of duodenal ulcer. In other organs (intestine, liver and pancreas) the hypersecretion of ferments is generally the expression of beginning lesions, and has much less semiological value.

As examples of secretory alterations due to hypofunction, we can mention gastric hyposecretion with hypochlorhydria and anachlorhydria. This situation is observed in chronic atrophic gastritis and in genuine Addison Biermer pernicious anemia.

SENSORY ALTERATIONS

They include pain itself and other imprecise sensory alterations or unpleasant sensations that patients refer to the abdomen. They are fundamentally epigastric pain, non-epigastric abdominal pain (from another location) and sensory neuroses, described by the German school.

We will see the pathophysiology later when studying abdominal pain. Likewise, it is essential to take into account, when assessing these alterations, the importance of other factors: the individual threshold for pain perception, the importance of the abdominal wall as a starting point for painful reflexes, the existence of the so-called digestive areas of relative sensory silence, as well as the enormous influence of the psychic factor with its vegetative connections, as we pointed out in the study of motor and secretory alterations.

As examples of sensory alterations we mention epigastric pain in duodenal ulcer, epigastric pain radiating to the right upper quadrant in cholecystopathies, non-epigastric abdominal pain in the left iliac fossa in cancer of the descending colon, etc., all of which will be studied in detail at the appropriate time.

MIXED ALTERATIONS

Motor, secretory and sensory alterations in fact coincide in every pathophysiological process, and in turn are seen in all possible combinations, hence the complexity of this aspect of digestive pathology. On the other hand, the pathogenic mechanisms are also multiple and converge irregularly in each case, confirming the difficulty in the semiological interpretation and diagnosis of this type of morbid alterations.

A typical example of the complexity of digestive pathophysiology is irritable bowel syndrome, in which there are predominantly motor alterations, but there are also secretory alterations and sensory alterations. In the etiopathogenic order, there are many factors that have been pointed out: dietary fiber, factor-psychosocial res, alterations in colonic motility, alterations in visceral perception, among others.

DISORDERS IN MASTICATION, SWALLOWING AND SALIVAL SECRETION

In general pathophysiology, these functional alterations must also be considered, although of less importance in their semiological application, when compared to the alterations previously studied. The concept, until now well-established, of the influence of masticatory deficit on digestive functions is discussed, and the dyspepsias due to lack of chewing cited in classic treatises (Fletcher) are questioned. On the contrary, excessive chewing is considered by some contemporary authors as a cause of faster gastric evacuation and, therefore, as a previous step to a more embarrassing intestinal digestion. Regarding the functional alterations of swallowing and salivary secretion, we refer the reader, so as not to fall into sterile repetitions, to Chapter 56, where the symptom dysphagia and the symptoms sialorrhea and xerostomia are discussed, since this knowledge has a more particular character.

MEDICAL RECORD

As we well know from general semiology, the protocol or clinical history is a document that contains the complete clinical study of a given patient, and in which there is a compilation of all the useful data related to the diagnosis, prognosis and treatment of each case. This document, in turn, is archived for teaching purposes, either directly or through medical publications.

In the semiological order we limit ourselves, within the protocol, to the study of each and every one of the subjective and objective symptoms that are grouped for this purpose, following the classic anatomical segmentation: from the mouth to the cardia, from the cardia to the ileocecal valve, and from this to the anus, with the aim of making this symptomatic grouping as didactic as possible. We will also study the laboratory and instrumental exams. We are now going to discuss in a general way the comparative assessment of these different methods in their specific application to gastroenterological diagnosis. They are:

-Anamnesis.

- Physical examination.
- Biochemical-clinical examinations and functional examination test (laboratory).
- Imaging examination.

Endoscopic examinations of the digestive tract and abdominal cavity.

Histopathological examination using biopsies.

Other special tests.

ANAMNESIS

The anamnesis or questioning of patients with the digestive system is a valuable diagnostic method. There are so-called typical and atypical anamnesis. A typical anamnesis is one in which the patient relates with semiographic accuracy the illness he suffers from. A typical anamnesis in this semiological chapter corresponds, for example, to many cases of gallstones, in which the patient spontaneously reports with great detail and semiographic accuracy his hepatic cramps, followed by jaundice and fever to a variable degree, all with a relatively periodic evolution; The patient, in turn, describes the particular characteristics of the pain (location, irradiation, time, etc.) very clearly, in order to be classified as corresponding to this disease. Many cases of duodenal ulcer also offer a typical anamnesis; with hunger pain, late postprandial. On the contrary, in atypical anamnesis the symptoms are so irregular that it is impossible to fit them into the mold of any disease.

Between these extremes of typical and atypical anamnesis are the vast majority of anamnesis, which must be given greater care and analysis.

In the assessment of patients with digestive diseases we will see, as Moynihan said, that in digestive pathology the interrogation is almost everything and the physical examination is hardly anything.

The anamnesis or interrogation in digestive semiology, as in any other system, is fundamentally composed of the following aspects:

- Personal identity data.
- Reason for admission or consultation.
- History of the current illness.
- Personal pathological history.
- Family pathological history.
- Toxic habits.
- Psychosocial history.

We defend the criterion of preparing for the patient the history of the current illness with the reason for consultation or admission, prior to collecting the data corresponding to his previous personal and family pathological history, since the inversion of this order contravenes in practice the natural anxiety of the patient to communicate to the doctor in the first place what ails him.

Of the related aspects, we will only study personal identity data and toxic habits.

Personal identity data

We will refer to:

- Origin and nationality.
- Age.
- Sex.
- Profession.

Origin and nationality

It is important to consider the origin of the patients, with regard to the influence, especially of infectious and contagious and racial or nationality factors. For example, it is interesting to know the existence of epidemic and endemic areas, since the origin of the patients in them automatically implies the possible diagnosis of the diseases that exist there.

An example is the fact that when faced with cystic hepatomegaly we hesitate to diagnose hydatosis if the patient has never left Cuba, since this parasitic infection does not exist among us and, on the contrary, is common in Argentina.

In relation to nationality, it is also interesting to note that gallstone disease is common in the United States of America and Sweden, where more than 60% of the population suffers from it at some point in their lives, and on the contrary, it is exceptional in black Africans; It is probably due to the very different type of diet that exists between countries.

The very high frequency of stomach cancer has also been proven in Japan, Chile and Costa Rica, for example.

Age

The semiological value of age is inestimable. The digestive system reacts differently at each stage of life in relation to stimuli, susceptibility and the capacity of the immune response to different aggressive agents or genetics.

From this it follows that the evaluation in the newborn with jaundice is very different from the adult patient with an icteric syndrome.

Sex

The predisposition for sex is conditioned to the metabolic changes determined by the different genital crises that the human organism goes through throughout its life, well differentiated in men and women.

1. In women these events have much greater importance than in men, since their endocrine crises predispose them to different digestive diseases. Let us point out, first of all, gallstones, a disease most common in adulthood, but also possible in the climacteric and in some cases in puberty, always coinciding with moments of genital crisis (puberty, pregnancy, lactation, critical age).

Let us also point out the predisposition of women to functional digestive diseases of neurovegetative cause.

2. In men we observe, more frequently than in women, gastroduodenal ulcer, liver cirrhosis, especially in alcoholics, and esophageal cancer, which according to statistics predominates in men (82%), compared to women.

Profession

In relation to the subject's occupation we must mention the influence of different factors closely related to their work. Professions predispose to different digestive diseases due to the psychological alterations they imply; Gastroduodenal ulcer, irritable bowel syndrome is related to professions that are subject to stressful situations (company directors, some specialties of medicine, military, etc.). Health professionals are at special risk for contamination with the hepatitis B virus.

Sedentary professions predispose to chronic constipation and hepatobiliary conditions. Excessive working hours and physical exhaustion are also predisposing factors. Finally, there is a relatively specific digestive pathology in certain types of work, such as contact with metals (lead) in workers in printing presses, painters, glass factories, etc.

In reality, the predisposition of workers to these diseases due to specific work decreases significantly, as in our country, when there is the protection of social laws and the development of occupational medicine.

toxic habits

Due to the importance they have in digestive pathology, it must be taken into account that:

1. Cigars and tobacco. Smoking, both cigarettes and tobacco, increases the predisposition to gastritis and gastroduodenal ulcer, and makes them worse.
2. Alcohol. Alcoholic gastritis is known, which is characterized by pititus or esophageal regurgitation. Alcoholic hepatitis and certain types of cirrhosis may occur in chronic alcoholics.
3. Coffee. In our country, in patients who drink pure coffee several times a day on an empty stomach, feelings of heartburn, gastritis and dyskinesias with gastric hypersecretion are common.

PHYSICAL EXAMINATION

It is a method of much less value in this semiology. Even organic processes sometimes evolve without any physical expression and the physical examination begins to be useful as soon as grossly evolutionary anatomical alterations are established, which make obvious the objective symptoms that are collected by inspection, palpation, percussion and auscultation.

The virtue of the semiotician in the physical examination of the abdomen consists of knowing how to capture everything that can be captured with clinical value, not exaggerating virtuosity, since this paradoxically makes this method of exploration confusing and useless.

Without a doubt, we repeat, the physical examination is of comparatively less value in relation to the other resources of gastroenterological diagnosis, as well as it is inferior, when compared to the value it has in other systems (circulatory, respiratory, nervous).

COMPLEMENTARY INVESTIGATIONS

With the anamnesis and physical examination, methods called natural, classical, direct, clinical, etc., which began in the Hippocratic era (350 BC), we make the so-called provisional diagnosis, at the expense of which we guide the selection of complementary investigations to try to reach a definitive diagnosis, and they are the ones we list below:

- Laboratory, biochemical, microbiological, hematological, etc. tests.

Functional tests of different organs.

Imaging examinations.

Endoscopic examinations of the digestive tract and abdominal cavity.

Histopathological examinations through biopsies, and cytological examinations, which can be performed as a complement to endoscopic and imaging examinations.

DIGESTIVE SYSTEM. SYMPTOMS OF THE OROPHARYNGOESOPHAGEAL

56

Introduction

The study of the symptoms of the oropharyngoesophageal segment must be treated methodically and carefully, since it constitutes the cultural basis for training as a semiologist in this specialty of pathology. There are different bases for grouping these symptoms:

according to their semiographic importance, according to their frequency, even following an alphabetical order, as some authors do. We have decided to make an anatomically based grouping; that is, according to the digestive organ or segment in which the symptoms apparently originate.

We believe that this anatomically based classification is the most didactic, although we do not ignore its possible imperfection when we think that the origin of some symptoms in the different organs or anatomical segments of the digestive system is not exact. According to clinical anatomy and physiology, there are two anatomical segmentations: the classic and the so-called neoanatomical, which demonstrates, therefore, the complex relationship between the anatomical and the physiological and pathophysiological. We, taking advantage of the opportunity to make pertinent clarifications if necessary, are going to follow the symptomatic grouping derived from classical anatomical segmentation.

We will thus classify, for study, the subjective symptoms of the digestive system into three groups:

1. Symptoms of the oropharyngoesophageal segment.
2. Symptoms of the gastroduodenohepatobiliopancreatic segment.
3. Symptoms of the enterocollicorrectal segment.

In this chapter and in the following two, we will address the analytical study of each symptom, exposing in separate paragraphs the knowledge related to its concept, semiogenesis, semiography and semidiagnosis, as indicated in the introduction of this work.

This methodology seems indispensable to us for understanding the study of each symptom, a fundamental element to fulfill Coste's semiological aphorism of going from the symptom to the disease, and which constitutes, therefore, the basis of the diagnosis.

Symptoms that are related to the oropharyngoesophageal segment are:

Sialorrhea.

- Xerostomia.

-Lingual burning.

- Halitosis.

-Taste alterations.

- Bruxism.

- Dysphagia.

- Heartburn.

- Pituita.

Let us make some observations, by way of introduction, in relation to this group of symptoms. Sialorrhea and xerostomia correspond to alterations in salivary secretion, due to an increase or decrease respectively. Let us point out that these symptoms, studied now in their subjective expression, sometimes have their objective equivalent, which we collect in the physical examination of the patients. Burning tongue, halitosis and taste alterations are subjective symptoms corresponding to the mouth, although their genesis often depends on general or systemic causes.

Dysphagia, in its high and medium localization modality, is a symptom referred to the pathology of the esophagus itself, but this symptom in its low modality, strengthens its relationships with heartburn and the pituitary gland. These esophageal symptoms: lower dysphagia, heartburn and pituitary, have, in turn, a very close pathophysiological relationship with gastric regurgitation and aerophagia, gastric symptoms dependent on alterations of the upper portions of the stomach, which is why they can be classified as a whole in the neoanatomical sense as being of juxtacardial origin.

SIALORRHEA

CONCEPT

From the Greek sialon: saliva; rhein: run. It is defined as the subjective sensation corresponding to the obvious increase in saliva. At certain moments the mouth is perceived to be full with saliva and, sometimes inevitably, the need to spit or swallow constantly;

On other occasions, the passive release of saliva to the outside, sliding at the corners of the mouth (generally only through one of them), thus determines, through its objectification, the easy identification of this symptom. The thread of saliva hanging from one of the corners of the mouth in an old, cachectic-looking patient, You have Roger's sign, corresponding to esophageal cancer (pathological esophagosalival reflex).

The synonymy of the sialorrhea symptom is rich and of variable interpretation. The common people use the term salivation and some authors prefer the medical term ptyalism. The term salivary hypersecretion is also equivalent. If the saliva is swallowed, it is called sialophagia. There is no sialorrhea without ptyalism or flow of saliva to the outside, since constant sialophagia is very painful for the patient; However, ptyalism is possible without true salivary hypersecretion in cases of swallowing paralysis and in psychopaths (Parmentier and Chabrol). Any salivary hypersecretion above 2 L in 24 h can be considered pathological, since the normal amount is 1,200-1,500 mL/day.

SEMIOGENESIS OR PHYSIOPATHOLOGY

The most accepted pathophysiological mechanism of sialorrhea revolves around the following factors:

-Excitability of the salivary reflex (neural mechanism).

Irritability of the salivary glands themselves.

Salivary reflex excitability

The stimulus that initiates the reflex can be located in the salivary glands or in their vicinity (at any point in the oral cavity) outside the mouth (at any point in the digestive system or even outside it), at a greater or lesser distance, and act as an " irritative thorn" giving rise in the pathophysiological order to an abnormal salivary reflex. If this reflex causes excitation of the glandular cells, the response will be an increase in salivary secretion, which constitutes the most frequent semiogenesis of the sialorrhea symptom.

It is important to note that if the irritating spine or site of birth of the reflex is located a short distance from the salivary glands, considered in this case as effector organs, the response will be more evident, easier and more intense; and on the contrary, if the irritating thorn is located at a great distance from the salivary glands, the response will be difficult and poor. This constitutes what, from this moment on, for purposes of future application in the semiogenesis of other symptoms of a reflex neural nature, we will call the law of the distance of the irritating spines, which we will cite on many occasions in connection with the study of other similar pathophysiological mechanisms in symptoms considered to be of a reflex type. This explains why an organic lesion, wherever it is found, can trigger a reflex of salivary excitement or sialorrhea; but in general it will be poor if said irritating thorn is at a distance, a salpingitis, for example.

On the other hand, any lesion located in the mouth, a simple tooth extraction, will cause a marked excitation reflex, which causes intense sialorrhea.

The above explains the multiple possibilities of sialorrhea by reflex mechanism, from an oral lesion to a more or less distant peripheral alteration including an alteration of the cerebral cortex, even of a functional or psychological nature. The increase in salivary secretion due to sensory stimuli, the sight of pleasant foods and even due to psychic stimuli and conditioned reflexes is well known (remember Pavlov's famous experiment). Let us point out, as another physiological fact of important semiological application that is accepted, the parasympathetic action, predominantly as excitation by vasodilation, and the sympathetic action, predominantly as inhibition by vasoconstriction, although the respective actions of the opposite type exist to a lesser degree.

Sialorrhea, therefore, should be considered as an expression of a predominant excitation of the parasympathetic system. (Experimental verification of sialorrhea due to excitation of the chorda tympani, which represents the parasympathetic nervous branch, is evident.) The influence of the neurogenic salivary reflex factor on the semiogenesis of sialorrhea is thus explained in general terms. We should point out that there may also be sialorrhea due to direct pathological excitation of the bulbar salivary centers, caused by hormonal and humoral alterations (exogenous and endogenous toxins circulating in the blood) (fig. 56.1).

Irritability of the salivary glands themselves

Any beginning histological alteration of the glandular cells will paradoxically determine hypersecretion and, consequently, sialorrhea. The progressive evolution of the lesions towards destruction will, on the contrary, produce xerostomia, as we will see in detail when studying this symptom.

SEMIODIAGNOSIS

We will present the semiological significance according to the distance of the irritating spines from the effector organ, going from the description of the oral processes to those located in the most distant organs and systems (fig. 56.2).

1. Sialorrhea of digestive cause. It can be seen in:

   a) Processes that affect the oral cavity. Within the oral cavity, we can mention inflammatory processes in the mouth (stomatitis), tooth extractions, etc.

   b) Processes that affect the salivary glands. When they are of low intensity or in their initial phases they produce sialorrhea; Epidemic parotitis can be mentioned in this sense, as well as some toxins (iodine) that directly affect these glands.

   c) Localization processes in the esophagus. Sialorrhea is a frequent symptom in low-lying esophageal processes, especially in esophageal cancer, a semiographic fact that, due to its clinical importance, was pointed out when defining this symptom. We also refer to sialorrhea caused by foreign bodies in the esophagus and by juxtacardial esophagitis of any origin. In some of these cases, sialorrhea becomes very intense, with a secretion of 3.5 and up to 10 L being observed, which can disturb the patient's sleep due to the associated sialophagia and ptyalism.

   d) Processes located in other segments of the digestive system. Sialorrhea corresponding to other digestive segments is also a symptom of gastritis, duodenitis and some types of gastroduodenal ulcers, particularly accompanied by marked vagotonia. We have observed sialorrhea as a symptom suggestive of pyloric hypertrophies in adults. The presence of sialorrhea is also described in pancreatitis, which is why we must point out the marked histological analogy between the pancreas and the salivary glands. Within the digestive system, finally, reflex sialorrhea is sometimes observed in certain types of colitis and appendicitis.

2. Sialorrhea of extradigestive cause. Sialorrhea is observed in the processes of the genital area of women. Saliveo or sialorrhea in pregnant women is typical, where this symptom acquires the category of an early sign of pregnancy, pointed out by the relatives of pregnant newlyweds. Sialorrhea during pregnancy is explained by both a reflex and hormonal mechanism. Let us point out the sialorea observed in cases of trigeminal neuralgia; Of interest is the sialorrhea corresponding to Pavlov's conditioned reflexes of a pathological type, observed in many types of psychoneuroses, carriers of the so-called nervous dyspepsias or dyskinesias. In other processes of the nervous system, such as Parkinson's disease, sialorrhea is observed. In hemiplegia, ptyalism or passive exit of saliva out of the mouth due to muscle paralysis (spurious or false sialorrhea of Von Bergman and Gigon) is rather appreciated. Let us also mention drug-related sialorrhea, due to pilocarpine, jaborandi, digitalis, etc.

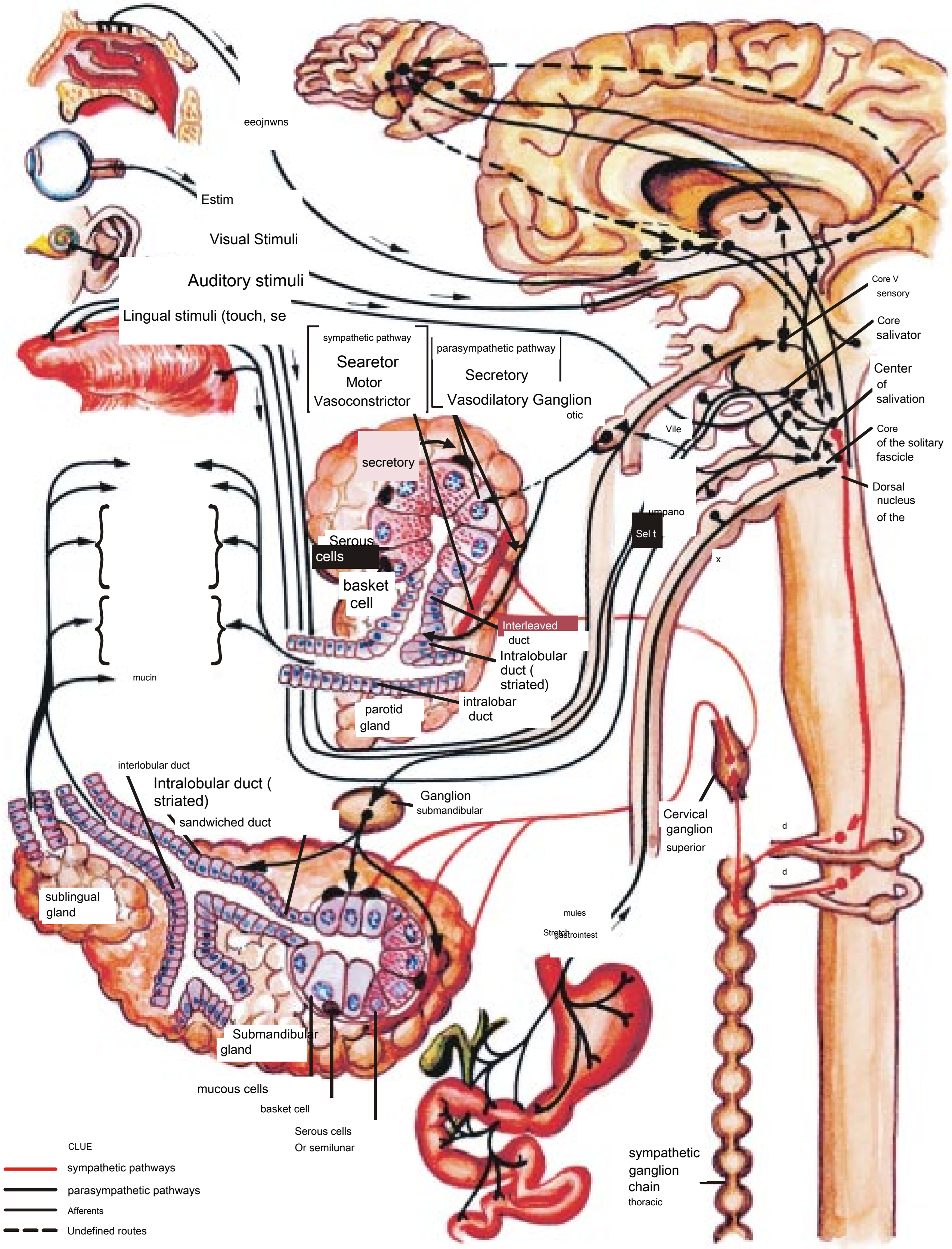

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Fig. 56.1 Semiogenesis or pathophysiology of sialorrhea.

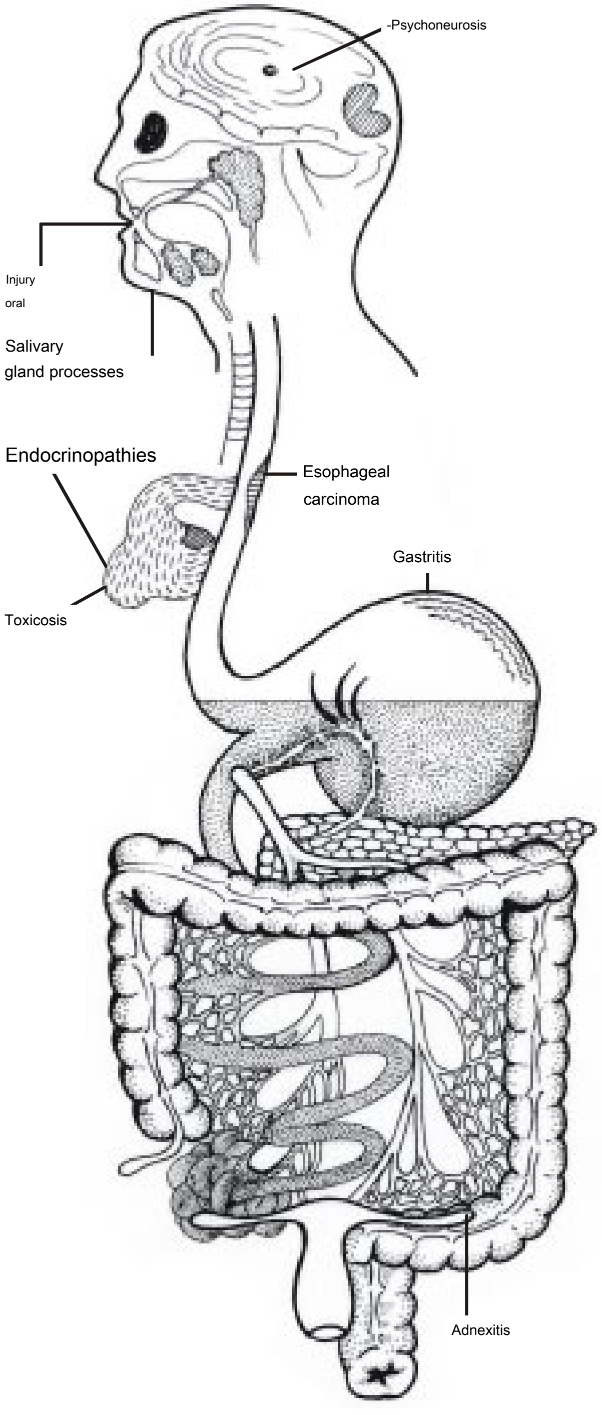


Fig. 56.2 Semidiagnosis of sialorrhea.

XEROSTOMY

CONCEPT

From the Greek xeros: dry; stoma: mouth. It is defined as the subjective sensation corresponding to the decrease in saliva; the patient perceives the sensation of dry mouth.

The identification of xerostomia is facilitated by the objective verification of an opaque tongue, like toast, a finding that is studied in the physical examination. Salivary hyposecretion is considered pathological when it is less than 0.5 L/day.

SEMIOGENESIS OR PHYSIOPATHOLOGY

We must consider the following factors:

1. Inhibition of the salivary reflex (neural mechanism). It is the opposite pathophysiological phenomenon to that which produces sialorrhea. Any process capable of reducing or abolishing the salivary reflex will cause xerostomia.

   As an example we can point out emotional psychic alterations (disgusts, joys, scares, etc.).

   It is accepted that in xerostomia there is a predominance of sympathetic action with vasoconstriction, which inhibits the secretion of the parotid gland and then the submandibular gland.
2. Some drugs can also produce it through their action on the nervous centers.
3. Destruction of glandular cells. The progressive destruction of glandular cells is logically responsible for xerostomia whose evolution will be parallel to the degree of cellular destruction, and may be reversible if anatomical reintegration is established.
4. Obstruction of the excretory ducts. Obstruction of the Stenon, Wharton, Rivinus and accessory ducts causes xerostomia, although the glands are not originally affected.
5. Decreased blood flow. Finally, in the semiogenesis of xerostomia, the decrease or absence of the water factor due to dehydrating processes of any nature is very important, even if there is anatomical integrity of the glands and their excretory ducts, and a normal salivary reflex.

SEMIOTECHNICS

Simple inspection of the oral cavity will show a dry, toasted tongue, and palpation will reveal the characteristic dryness and roughness.

SEMIODIAGNOSIS

Xerostomia can be caused by:

1. Inhibition of the salivary reflex. It is relatively rare when compared to sialorrhea, determined by the opposite phenomenon. However, let us point out the accidental xerostomia of emotional subjects and psychoneurotics (anxiety, anguish, fear syndromes).
2. Destruction of the glands. It is seen in Mikulicz disease and Sjögren's syndrome.

(certain types of pansalival lymphogranulomatosis), in salivary gland tumors of different types; In all these processes, a progressive destruction of glandular cells is established.

3. Obstruction of the excretory ducts. In these cases it is accompanied by local pain, which is exacerbated by some foods. Let us mention the xerostomia that is observed in salivary lithiasis (sialodochitis).
4. Decrease in blood flow from dehydrating processes. Atropine poisoning can be cited as an example of drug-induced xerostomia.

LINGUAL OR BUCOLINGUAL BURNING

CONCEPT

Let's define this symptom as the burning sensation, sometimes even burning, that some patients experience in their mouth and particularly on their tongue.

Patients identify this symptom by its greater location on the tip and edges of the tongue. It is produced or exacerbated by contact with foods loaded with salt, spicy, very cold or hot. Sometimes, buccolingual burning becomes constant. When the sensation is frankly painful, we speak of glossodynia or glossalgi.

SEMIOGENESIS OR PHYSIOPATHOLOGY

It occurs as a result of neural irritation of the buccal and lingual mucosa. In all cases there is a more or less marked inflammatory alteration of the lingual papillae, with hypertrophy, atrophy or both processes at the same time. Neural irritation with papillary alteration can be produced mainly by the following factors:

1. Nutritional deficiency. It is selective in nature; B complex deficiency (particularly niacin and riboflavin), iron deficiency, and protein deficiency are noted as the most frequent factors.
2. Digestive processes. Those that involve alterations in gastric acidity and stagnation at different levels of intestinal contents, accompanied by retroperistalsis, such as colonic ectasia, etc.
3. Elimination of toxic substances. When carried out by the oral mucosa.
4. Organic lesions of the mouth. Which act by direct neural irritation.
5. Idiopathic or essential.

SEMIODIAGNOSTIC0

There are multiple diseases in which this symptom is present, among the most important we have:

1. Nutritional deficiency diseases in which chronic glossitis, more or less intermittent, runs parallel to diarrhea and anemia, causing glossoenteroanemic syndrome. The most typical example of a disease that causes burning tongue is severe sprue, which justified the name "painful mouth of Ceylon" to describe these syndromes in India.

   Let us mention here the burning tongue, more discreet by the way, of genuine pernicious anemia, of Addison Biermer, with tongue atrophy and aquilia (Moller-Hunter glossitis).
2. Digestive diseases themselves: gastric dyspepsia, either with hyperchlorhydria or, more frequently, with hypochlorhydria and anachlorhydria.
3. Intoxications, in which there is oral elimination of metalloids.
4. Local diseases of the mouth and tongue with predominant glossitis, such as leukoplakia and lichen planus.
5. Finally, it is interesting to mention idiopathic glossodynia or painful burning of the tongue, without obvious cause, evident in psychopaths and psychoneurotics.

HALITOSIS

CONCEPT

Normal breath is odorless, it is altered only circumstantially and temporarily (food, foreign substances, age, sex, fasting, etc.). The bad odor or stench of the breath, more or less permanent, is a pathological phenomenon, this is how halitosis is defined, a symptom whose medical and social importance is extraordinary. Sometimes patients who know the existence of their halitosis have greater defense, but those who ignore it or hide it, to their own detriment, have social and sexual difficulties.

Let us point out, however, that due to psychoneurotic or psychotic alteration, some subjects falsely insist on believing that they have halitosis, for which reason its existence should always be verified by the doctor.

SEMIOGENESIS OR PHYSIOPATHOLOGY

We will describe four fundamental mechanisms:

1. Halitosis of local cause, produced by fetid foci in the mouth and its dependencies.
2. Halitosis of respiratory cause, produced by fetid processes in the bronchi and lungs, in which the exhaled air is impregnated with deleterious materials from the sources of suppuration.

3. Halitosis of digestive cause, properly speaking, produced by reversal peristalsis or antiperistalsis phenomena, well described by Álvarez. In these cases, a retrograde circulation of gases is established from different levels of the digestive tract, which are deposited in solid form at the base of the tongue, determining halitosis.

   Kast, through experiments on patients, has been able to verify these phenomena of reversal peristalsis, common in the production of other digestive symptoms, observing in the mouths of said subjects lycopodium powders deposited in distant parts of the intestine (in the rectum and in the sigmoid, which could be done by means of a rectosigmoidoscopic tube).
4. Halitosis caused by blood, due to elimination at the level of the lung alveoli and incorporation into the expired air of abnormal deleterious substances circulating in the blood.

SEMIOTECHNICS

We can check it by smelling the patient's breath.

Lermoyez has pointed out some very useful rules, which we reproduce below:

1. If the air exhaled through the nose is foul-smelling and the air exhaled through the mouth is not, we must think that the cause is above the soft palate (rhinitis, especially atrophic rhinitis, sinusitis, etc.).
2. If the air exhaled through the mouth is fetid and the air exhaled through the nose is very little, the cause lies in the mouth: cavities, pyorrhea, tartar, etc.
3. If the two exhaled airs are equally fetid, the cause lies in the pharynx (chronic pharyngitis, tonsillitis), in the respiratory system (abscesses, bronchiectasis); in the digestive system; in general diseases (such as uremia) or in poisoning.

SEMIODIAGNOSIS

We will briefly list different diseases capable of producing halitosis, following in parallel the same order set out in semiogenesis (fig. 56.3):

1. Among the local fetid diseases of the mouth we point out dentures in poor condition, cryptic tonsillitis, etc. According to Chiesa, 90% of foul breath is of oral origin.

2. Halitosis can be due to nasal lesions: rhinitis, adenoiditis, sinusitis.
3. In the group of lesions of the respiratory system we mainly have bronchiectasis and pulmonary gangrene.
4. Among digestive diseases with halitosis we can mention esophageal diverticula and pyloric obstruction (caused by fermentation of retained foods), constipation, etc.

5. Halitosis due to respiratory elimination from the blood is observed in some types of menstrual disorders, and is markedly exacerbated in the days surrounding menstruation itself. This modality has, as we said before, great social and psychosexual importance, and sometimes causes states of psychoneurosis with an inferiority complex. In addition to this halitosis typical of women, let us mention to finish this group, the halitosis of diabetes, uremia and acute liver necrosis.

   In the latter, the breath, which is actually sweet and special, has a high diagnostic value. Drug and tobacco poisoning are of complementary semiological value, since they occur in parallel with the rest of the symptomatology of said processes.

TASTE ALTERATIONS

CONCEPT

They are defined as the subjective sensations of bad taste that some patients perceive in their mouth and tongue, and which they report with some frequency in the anamnesis. Patients mainly identify it as bitter, sour, sweet, pasty and metallic.

They can also refer to the loss or absence of taste (ageusia) , as well as its perversion (dysgeusia, parageusia, or cacogeusia); These last alterations mentioned are more typical of neuropsychic pathology.

SEMIOGENESIS OR PHYSIOPATHOLOGY

Taste alterations correspond, in a general order, to the same causes of the halitosis symptom that we have just described. We must consider in parallel the local causes, respiratory type, digestive type and blood type or elimination.

Within the digestive group, we should point out in particular that the antiperistaltic phenomena that produce them are relatively different. The bitter taste is supposed to be linked to biliary alteration due to reflux, although the importance of the psychological factor is also noted. The sour taste corresponds to phenomena of gastric regurgitation, generally of hyperacid content. The sweet taste is hypothetically supposed to be related to antiperistalsis phenomena of intestinal origin. The pasty taste seems to coincide with the so-called coated tongue, determined by an immobilization of the filiform papillae and

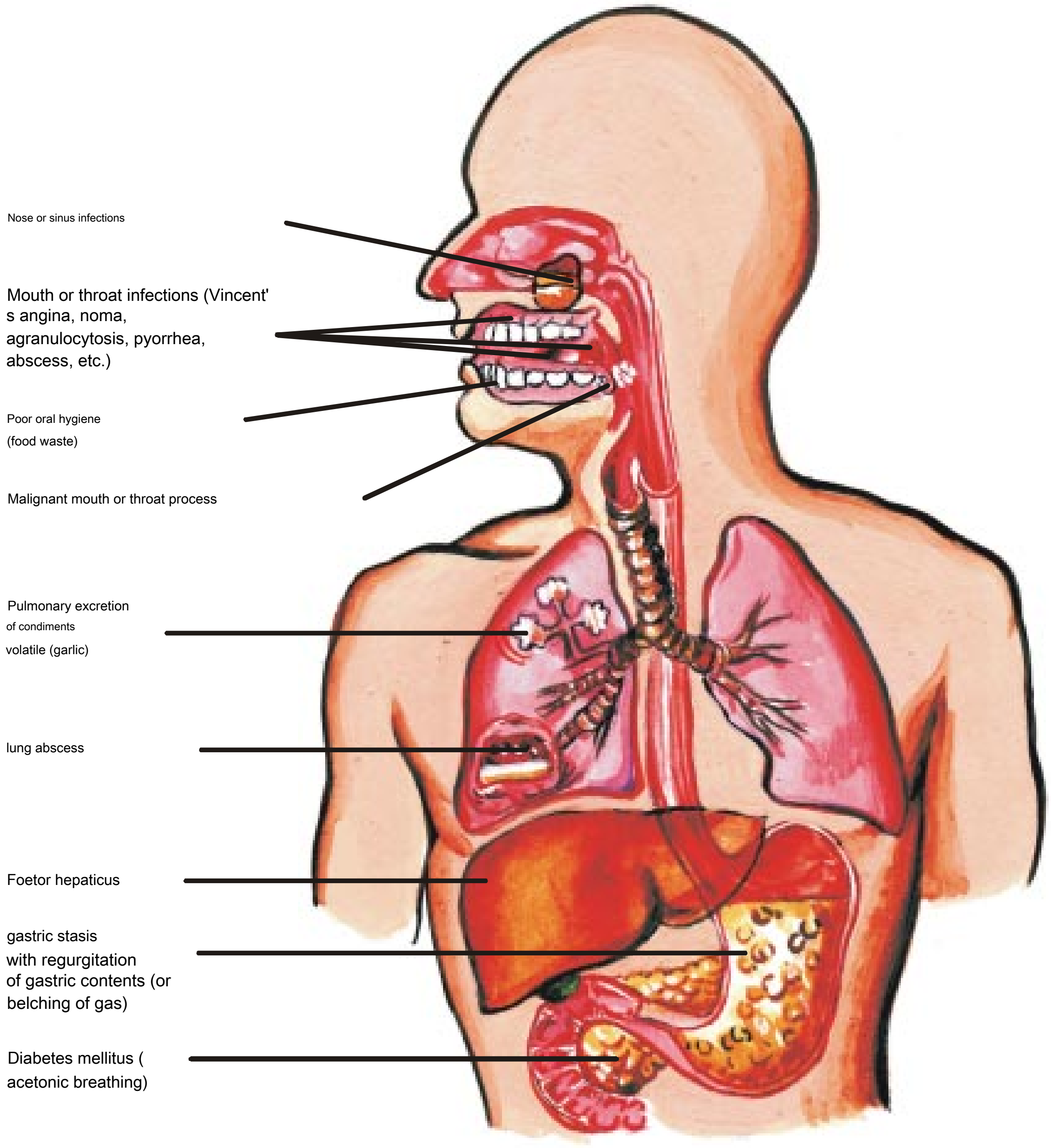

Nose or sinus infections
Mouth or throat infections (Vincent' s angina, noma, agranulocytosis, pyorrhea, abscess, etc.)
Poor oral hygiene (food waste)
Malignant mouth or throat process
Pulmonary excretion of condiments volatile (garlic)
lung abscess
Foetor hepaticus
gastric stasis with regurgitation of gastric contents (or belching of gas)
Diabetes mellitus (acetonic breathing)

Fig. 56.3 Semidiagnosis of halitosis.

whose objective alteration is studied in Chapter 59. The metallic taste, present in some dyskinesias, is also observed due to local causes in subjects wearing dental plates or other prostheses, although it is also produced by other oral pathological processes, such as suppurative foci of the tonsils, pharynx, etc.

Ageusia and dysgeusia have as their mechanism the neural alteration of the taste buds. We must remember as an anatomical fact of interest, the particular lingual location in the perception of flavors: bitter is located at the base of the tongue; the sour at the edges, and the sweet and salty at the tip. As for the

Causes -local, respiratory and blood- are similar to those indicated in halitosis.

SEMIODIAGNOSIS

It is an obligatory corollary of what was stated above. The bitter taste, which some authors refer to as a constant discomfort, is observed in chronic functional and organic hepatobiliary processes. It is also seen in nervous dyspeptics without manifest biliary alteration. The sour taste is observed in some cases of gastric dyskinesia, gastritis with hyperchlorhydria and duodenal ulcer.

Sweetish, pasty and metallic flavors are observed in some dyskinetic gastrointestinal processes, as well as in cases of tonsillitis, dental caries, dental prostheses, nasopharyngitis, etc. Blood or elimination taste alterations are also observed in some ovarian-type endocrinopathy, menstrual disturbances, as well as in endogenous and exogenous poisoning, and in the administration of some medications (iodides, etc.).

Ageusia and dysgeusia are observed in paralysis of the gustatory nerves (the lingual, glossopharyngeal and chorda tympani, mainly) and in psychoneuroses and psychopathies; They constitute elements of semiological value in diseases of the nervous system.

BRUXISM

Also called teeth grinding. This is understood as the circular movement of the teeth in the direction and lateral direction of friction in the inclined intercuspal planes, with intense pressure and which is sometimes uninterrupted during the night. This joint pathological mechanism, called by some brichism, brichomania (from the Greek, brychein: grinding of teeth) or bruxomania, is always accompanied by a characteristic sign, which consists of the intense and prolonged contraction of the masseter muscles during sleep, which causes continuous pressure on its superficial and deep fibers. When it occurs at night it is called the Karolyi phenomenon.

It is observed in nervous and neurotic subjects, as well as in intestinal parasitosis and latent tetany (Hufschmidt-Heim).

DYSPHAGIA

Before defining and studying this symptom, it is advisable to briefly review the anatomy and physiology of the esophagus and swallowing.

ANATOMICAL SUMMARY OF THE ESOPHAGUS

The esophagus is a musculomembranous tube that runs from the hypopharynx to the stomach. Its average length is, according to Palmer, 25 cm in men and 23 cm in women, with possible variations of 6-9 cm. The distance from the incisors to the cardia is 40 cm on average in men, and 37 cm in women (Leriche). The diameters are 1.3 cm at the level of the cricoid cartilage; 1.3 cm at the tracheal bifurcation; 1.95 cm at the diaphragmatic orifice, and 2.2 cm at the level of the esophagogastric junction. the mouth Superior, or Killian's mouth, is located at the level of the lower edge of the cricoid cartilage and C6. The esophagus extends along the anterior surface of the spine, adapting to its anatomical curvatures. It crosses the diaphragm at the level of D11 and passes into the abdominal cavity, where it empties into the great tuberosity of the stomach.

It has three portions: cervical, thoracic and abdominal; and four normal narrowings: mouth-esophageal, aortic (at the level of D4), bronchial (at the level of D5) and diaphragmatic (at the level of D11).

The cervical portion is in relation: in front, with the trachea; at the base of the neck, with the left lobe of the thyroid body0; behind, with the cervical spine; on each side, with the common carotid and partially with the thyroid gland. Recurrent nerves circulate between the esophagus and the trachea.

The thoracic portion, initially located in the upper mediastinum, between the trachea in front and the spine behind, descends to the posterior mediastinum, passing backwards and to the right of the aortic arch, behind the left bronchus. It immediately extends along the right edge of the descending aorta, then passes in front and to its left, and enters the abdominal cavity through the diaphragmatic orifice. Thus, the anterior surface is in relation to the trachea, the origin of the left bronchus, the pericardium and the diaphragm. The posterior surface is in contact with the vertebral column, the right intercostal arteries and the small azygos vein. To its left is the arch of the aorta, the left pleura, and then the descending aorta; to its right, the right pleura and the great azygos vein. Below the pulmonary hilum the pneumogastric nerves descend, in contact with the esophagus; the left, in front, the right, behind. The branches of the two nerves join to form a plexXO.

The abdominal portion includes the esophageal length in relation to the posterior wall of the left lobe of the liver; The anteroexternal surface is covered by the peritoneum. The length of the abdominal segment is variable from one individual to another. Leriche, with the name of gastroesophageal vestibule, and Nemours-Auguste, with the name of epicardias, have described a region limited by two constriction rings: the lower esophageal sphincter and the constrictor of the cardia. Leriche has insisted on the presence of a phrenoesophageal elastic membrane that extends behind the esophageal wall up to 2 or 3 cm above the diaphragm and that prevents excessive shortening due to the contraction of the longitudinal muscles and intra-abdominal pressure.

Laimer's membrane has been described as a fibrous sheath that forms an envelope into which the esophagus is inserted. Ensures a tight separation between the abdominal and thoracic cavities and is confused with the periesophageal tissues, above the diaphragm, and with the peritoneum, below it.

The arteries come from the inferior thyroid artery, the aortic arch of the descending aorta, the left gastric branch of the celiac artery, and the left inferior phrenic branch of the abdominal aorta. Generally, its direction is parallel to the great axis of the esophagus.

The veins that come from the upper half of the esophagus meet in the inferior thyroid vein, the azygos vein, and the hemiazygos vein. Those that come from the lower half flow into the splenic and gastric veins.
They constitute a place particularly exposed to the formation of varicose veins.

The nerves that innervate the striated muscle fibers come from the recurrent nerves. Those that innervate the smooth fibers come from the parasympathetic and sympathetic trunks, and their endings form a plexus between the two muscle layers and the submucosa.

The tracheobronchial nodes form four main groups: tracheal group on each side of the trachea, tracheobronchial group at the angle of the tracheal bifurcation and at the origin of the main bronchi, which is in direct relation to the esophagus; bronchopulmonary group and pulmonary group.

HISTOLOGICAL SUMMARY OF THE ESOPHAGUS

The mucosa of the esophagus, pink above, pale below, is a thick tunic arranged in longitudinal folds that are erased upon distention.
It is covered with a squamous, stratified epithelium, full of small papillae. Its base is formed by a layer of cylindrical cells that rests on the mucous membrane. In the subdiaphragmatic part, the epithelium acquires a cylindrical appearance, like the gastric one. The boundary between these two structures is highly disputed and probably variable. On the other hand, islands of gastric mucosa may exist in the esophageal mucosa.

The muscularis nucosae is absent in the upper part.

The subnucosa, or areolar layer, covered with connective tissue, joins the mucosa to the muscularis. Contains nervous fillet vessels and mixed mucous glands: tubular OR pure mucous, tubuloacinous.

The muscular layer is composed of two very thick layers: the internal one, of circular fibers, and the external one, of longitudinal fibers. In the upper part, at the level of Killian's mouth, the internal muscle fibers continue with those of the constrictor of the pharynx, forming a true sphincter. In the supra-aortic and retrocardiac segments, the muscle fibers of the inner layer have an elliptical shape and an oblique arrangement. At the level of the trachea they are circular, to become spiral in the juxtaphrenic segments (Leriche). The external longitudinal fibers form three fascicles in the upper region: one anterior, linked to the cricoid cartilage;
two lateral ones, which continue with the pharyngeal muscle fibers when they reach the base; The three muscular bands widen, contact and intersect, forming a uniform layer that covers the entire external surface.

The numerous striated muscle fibers in the upper third progressively decrease and practically disappear in the lower third.

Accessory fascicles start from the muscularis and cross the fibrous tunic to attach to the neighboring organs: pleura, aorta, left bronchus and pericardium.

The fibrous outer tunic has no individuality; is confused with that of neighboring organs. The cervical and thoracic portions of the esophagus lack serosa.

PHYSIOLOGICAL SUMMARY OF SWALLOWING

The swallowing mechanism occurs in three phases.

ses:

Voluntary phase. Contraction of the muscles of the tongue that evacuate the bolus towards the pharynx.

Involuntary and reflex phase. It carries the bolus through the pharynx, through the action of the pharyngeal constrictor muscles. These reflexes are triggered by tactile excitations of the base of the tongue, the tonsils, the soft palate and the posterior wall of the pharynx. The main innervation belongs to the glossopharyngeal, with some small terminal branches of the trigeminal (velum) and superior laryngeal (epiglottis).

The elevation of the dorsum of the tongue and the contraction of the pillars prevent its return towards the oral cavity, while the elevation of the velum prevents reflux towards the nasopharynx, and that of the suprahyoid, towards the larynx and bronchi, with the simultaneous play of the epiglottis. Breathing is inhibited. Thus, the bolus disappears in the hypopharynx, which rises; The cricopharyngeus muscle relaxes and the contraction reaches the upper esophagus where it becomes a primitive wave (Templeton).
Radiocinematography has made it possible to objectify these complex mechanisms.

Esophageal phase. The hypopharyngeal contraction continues in a primary wave without interruption along the esophagus with progressively decreasing amplitude. These afferent reflex actions are dependent on the pharyngeal branches of the glossopharyngeal, and the efferent influences are received by the vagus, but the intrinsic Auerbach plexuses play an important role in the propagation of this primitive wave. Local distention of the esophagus due to solid food or liquid pressure also produces secondary contractions that occur at the level of the aortic crossing and

They descend towards the lower part of the esophagus. Tertiary, irregular, non-peristaltic contractions are only seen in the lower esophagus: they are brief and spontaneous. They are attributable to the action of the intrinsic plexus, and disappear when a primary contraction passes. They are observed in elderly people or in certain pathological circumstances.

The esophageal path is, therefore, dependent on a pressure gradient produced mainly by primary peristaltic contraction; However, secondary contractions can be added to primary contractions if they are ineffective.

For liquids, whose travel is faster, especially in a vertical position, and exceed the speed of the primary contraction wave, the constrictors of the pharynx act as propellants. In the upright position, the liquid or semi-liquid esophageal passage generally lasts 5 s. It is slower in a horizontal position.

The musculature varies according to the sectors: the upper third is mostly provided with striated fibers; The middle third is made up of mixed striated and smooth fibers; the lower third contains nothing but smooth fibers. On the other hand, the upper striated portion does not contain intrinsic nerve fibers, while the lower two-thirds do.

The musculature consists of external longitudinal fibers, an internal tunic of circular fibers, also oblique or spiral, and a muscularis mucosae. Longitudinal fibers have the ability to shrink extraordinarily (55% of the initial length, according to Nauta).

For Nemours-Auguste, thoracic widening does not exist and there is no pharyngeal aspiration or shrinkage of the pharynx, but rather a massive elevation of the larynx-pharynx-esophagus. Almost all of the air is drawn during inspiration into the larynx and trachea, and a small portion enters the esophagus.

Innervation is provided mainly by the vagus. Its high and bilateral section causes paralysis of the body of the esophagus and distention of the distal segment, but this action is temporary and does not last more than a few weeks. Sympathetic innervation is only exerted at the level of the last centimeters; The fibers come from the celiac trunk and the endings of the left gastric arteries. The intrinsic nervous system only exists in the lower two thirds, especially the Auerbach plexus or myenteric plexus, which has motor activity. It is located between the internal and external muscular tunics and coils between the nerve cells forming ganglion groups.

The Auerbach plexus synapses with the terminal fibers of the vagus, but not with the simple nerve fibers. paths that pass through it without anastomosing with it. Vagotomy does not alter the cells of the Auerbach plexus, but its destruction causes the definitive suppression of peristalsis.

When it comes to the lower portion of the esophagus, there are conflicting opinions. The manometric and radiological data are also contradictory.

For Chevallier-Jackson, the closure of the lower portion of the esophagus is ensured by the muscular rings of the diaphragm (pincer action). Nauta by radiocinematography, demonstrated that the motor activity of the lower esophagus does not differ in any way from that of the rest of the organ and that there is no physiological sphincter,

Jutras and Nemours-Auguste considered that the epicardia acts as a true physiological sphincter:

A perfect rhythm can be seen by fluoroscopy, behaving as a true distributor." These rhythmic contractions are not synchronous with either the pulse or respiration. Fyke, Code and Schlegel have shown through manometric studies that there exists in the resting state a zone of elevated pressures that extends over 2 or 3 cm of the lower portion, and gives this region the physiological attributes of a sphincter. Garcin supposes that the epicardia It contracts the opening of the lower portion of the esophagus in the esophagogastric direction, while the cardia prevents gastroesophageal reflux. F. Besançon, Janin and Ch. Debray, through electromanometric studies, revealed a motor reaction of the body of the esophagus to distension in the form of a reactional cardiospasm.

With this prior knowledge we will now study dysphagia.

CONCEPT

Dysphagia is defined as the subjective sensation of difficulty swallowing, which is experienced temporarily or permanently.

As synonymous terms, patients speak of "stuckness", a sensation of "stuckness" of what they swallow, indicating this sensation at different levels of the esophagus. Some patients speak of a "ball" sensation when beginning to swallow. Sometimes the patient communicates the sensation of pain itself when swallowing, in which case we identify the symptom as odynophagia.

Sometimes, dysphagia is not subjective, but becomes evident when performing an esophagoscopic or radiological examination (objective dysphagia). Finally, it is interesting to note that sometimes imprecise subjective sensations of esophageal discomfort are identified, which are neither precisely dysphagia nor odynophagia, which are known as " motor and sensory neuroses of the esophagus" and which we must study in parallel to dysphagia.

SEMIOGENESIS OR PHYSIOPATHOLOGY

The difficulty in swallowing water, liquid or solid foods is generally due to either anatomical alterations of the oropharyngoesophageal duct or physiological alterations in swallowing. We can fundamentally point out, therefore, two pathogenic types of dysphagia: organic, mechanical dysphagia, with an anatomopathological basis, and functional, spasmodic dysphagia, with a pathophysiological basis. As we will explain later, both mechanisms can coexist in the same patient, and a more or less variable neighboring spasm must be accepted in all organic dysphagia. Let's expose both mechanisms.

Pathophysiology of organic dysphagia

In this type, the difficulty is easily explained because there is a decrease in oropharyngosophageal permeability. Any obstructive pathological process at any level of said duct occludes the lumen and causes organic or mechanical dysphagia. The occlusion can be intrinsic, caused by proliferative or stenosing processes that grow towards the lumen of the organ, or extrinsic, due to processes that grow and crush the esophagus from the mediastinum, also occluding its lumen.

It is important to point out, as we previously pointed out, that from the beginning of every obstructive organic process of the esophagus, and many times before said process, per se, mechanically occludes the lumen, a neighboring functional or spastic alteration is added, which is responsible for the occlusion or increases it. This functional factor can vary from one moment to the next or be compromised by the administration of antispasmodics or corticovegetative sedatives, which often explains the apparent improvement of an obstructive organic process.

Let's see below the hypothetical mechanism of said pathophysiological alteration.

Pathophysiology of functional dysphagia

The pathogenic mechanism of functional dysphagia is due to the multiple and complex alterations in the physiology of swallowing. Since this function is not exactly known, many hypotheses regarding this pathophysiological mechanism must be accepted.

Overall, we can say that this pathophysiology revolves around the alteration of the normal neuromotor coordination of swallowing. At the same time, the details vary relatively when we analyze in particular the pathophysiology of high, medium or low type functional dysphagia; that is, according to how it affects the different swallowing times.

High functional dysphagia

It is produced by alteration of the swallowing reflex with disorders of its centers or its afferent and efferent pathways. The multiplicity of pathological processes capable of producing functional dysphagia is easily understood. Particularly, any organic process close or distant to the esophagus acting as an irritative spine and conditioned to the law of the distances of irritating spines, cited in connection with the symptom sialorrhea, will be a cause of functional dysphagia. A psychological alteration may, through a similar mechanism, alter said reflex.

Paralysis of the swallowing nerves due to a bulbar lesion will determine functional dysphagia due to a central alteration.

When the lesions are located in the pharynx and at the entrance to the esophagus, the response will be localized reflexively in the upper parts, causing the high type of dysphagia.

Medium functional dysphagia

Any small intrinsic lesion, neighboring or distant from the esophagus, acting as an irritating thorn, may alter the relaxing peristalsis of this organ, determining a median localized dysphagia, which can be produced by reflex alteration or by alteration of the autonomic neuromuscular apparatus of the esophagus.

Low functional dysphagia

A low-lying irritating thorn will:

1. Alter relaxing peristalsis in the terminal phase and produce low functional dysphagia.
2. Produce a loss of vagosympathetic balance.
3. Alter the sphincters or their mechanical means of containment.

Following what was previously pointed out by Sánchez and Kramer, any motor alteration of the regions of the phrenic ampulla and the vestibule can also determine low functional dysphagia. Let us also point out, as another mechanism, the alteration of gastric acidity.

Finally, histological alterations of the different esophageal layers and the autochthonous neuromuscular system have been confirmed, which cast doubt on the pure functional nature of low functional dysphagia. In any case, the degenerative alteration of the Auerbach plexus, ganglion cells and nerve fibers pointed out by Etzel and others must be accepted as another mechanism of this type of functional dysphagia. Achalasia, as its name indicates, consists of a lack of relaxation of the cardia accompanied by an asynergy that would affect the propulsive mechanisms of the esophagus and the vestibule.

Although we have exposed the pathophysiological mechanisms of functional dysphagia, according to its high, medium or low location, in parallel to the three swallowing times, in the clinic these factors can overlap, producing variable and mixed types.

SEMIOGRAPHY

For the semiotician it is essential, whenever possible, to carry out an active, detailed interrogation, trying to identify the particular clinical characteristics of the symptom, which greatly facilitates successfully fulfilling the formula of going from the symptom to the disease."

In dysphagia it will be important to study the following characteristics:

Location

Dysphagia can be subjectively identified as high, medium or low in location.

High dysphagia is referred by the patient to the neck, corresponding to the projection of the cricoid cartilage (Killian's mouth or cricopharyngeal ring). The difficulty is perceived at the moment you begin to swallow. Let us mention among the organic oropharyngolaryngeal diseases, Zenker's cricopharyngeal diverticulum, some goiters, as well as inflammatory and allergic lesions of the region.

Within the functional type, the most frequent possibility is the so-called hysterical ball by ancient psychiatrists, which corresponds to a spastic functional dysphagia, of cortico-vegetative origin. Another type of high dysphagia is Plummer-Vinson syndrome, whose pathogenesis was initially considered by the authors to be of exclusively neurovegetative origin. This syndrome is due to a deficiency condition with idiopathic hypochromic anemia and anachlorhydria, glossitis, and there may be discrete inflammation of the upper esophagus and a ring may form that partially narrows the lumen. Let us also mention among the high functional dysphagias, that related to bulbar nerve palsies. Furthermore, let us remember that any injury (even if it is just beginning) to the pharynx or upper esophagus can produce this type of high functional dysphagia by acting as a reflexogenic spine.

Dysphagia of median location is referred by the patient to the thorax, corresponding to processes of the middle esophagus or this segment acting as an effector element. In these cases, the sensation of dysphagia is perceived 2 6 5 s after swallowing. The most frequent diseases of an organic nature are: carcinoma, scarring stenosis of the esophagus and Rokitansky traction diverticulum as intrinsic, and most extrinsic stenosis should also be considered, particularly aortic and mediastinal conditions of all kinds. In all these examples, as we have said, the neighboring spastic factor superimposed is common. dido. Diffuse spasm of the esophagus is worth noting as an example of a functional type.

Low dysphagia is the most interesting in the clinic because it exists in multiple diseases, both organic and functional. The patient refers it to the xiphoid appendage of the sternum and the highest portion of the epigastrium, perceiving a sensation of jamming between 5 and 15 seconds after swallowing. Among the organic diseases, the following are important: cancer of the lower esophagus and gastric fundus (juxtacardial), peptic ulcer of the esophagus, Grenet's epiphrenic diverticulum, foreign bodies and chronic lower esophagitis. They are important as functional types: transient spasm of the cardia and achalasia. Let us finally mention diaphragmatic hernias, so common, and juxtacardial gastric injuries.

Periodicity

This factor refers to the evolution of dysphagia over time from its onset. In general, periodic dysphagia that appears for certain periods of time, alternating with latency phases, corresponds to the functional type, and the presence of extraesophageal lesions and primary motor dysfunctions must be investigated. Let us point out as examples, cholecystopathies and transient spasm of the cardia.

Non-periodic dysphagias, that is, they follow a constant evolution since they are established, are usually of an organic nature, due to obstructive diseases of the esophagus, such as carcinoma, mediastinal tumors and caustic strictures.

Intensity

Dysphagia can be light, medium or marked intensity. In general, organic dysphagia reaches marked degrees, although it is not uncommon for some type of functional dysphagia due to motor impairment to also be intense. The beginning organic obstructions of the esophagus, as well as the processes of pure functional dysphagia, for the most part, are of light or medium intensity.

Quality

In this case we are referring to the relationship between dysphagia and the degree of consistency of the bolus when swallowing; that is, the behavior of the dysphagic patient when swallowing liquids, semi-solid and solid foods. Patients with organic dysphagia, for the most part, first observe difficulty swallowing solids, then semi-solid foods, and finally liquids, a fact that is recorded in the anamnesis of most cases of obstructive cancer, stenosing esophagitis, and mediastinal tumors. In cases of functional dysphagia, behavior is often observed paradoxical; That is, a patient with cardiospasm, for example, may notice difficulty swallowing a mouthful of water and yet swallow a piece of meat more easily.

Start mode

It can be abrupt, sudden or insidious, and slow. A sudden, abrupt onset may be due to the ingestion of a foreign body or the ingestion of caustic, organic dysphagia processes; But except for these examples, in general the abrupt onset corresponds to processes of functional dysphagia of vegetative cause, the hysterical ball" corresponding to the transient cricopharyngeal spasm, generally, has an abrupt onset.

Allergic processes, reflex spasms and achalasia all produce dysphagia of more or less abrupt onset. An insidious, slow onset generally corresponds to organic dysphagia processes, in which obstruction is established in an insidious manner at its beginnings. As an example of dysphagia of insidious onset, let us cite intraesophageal and extraesophageal malignant tumors.

Associated symptoms

Sometimes dysphagia is accompanied by:

a) Pain or odynophagia, in these cases we will always think of organic processes, especially malignant tumors and esophagitis, in which there is irritation of the nerve fillets. Esophageal pain is located retrosternally and can radiate to the neck, nape of the neck, and interscapular region.

b) Dysphagia is also sometimes associated with small hematemesis, an expression of hemorrhagic lesions of the esophagus.

c) In some cases, the presence of a rich array of psychovegetative symptoms is noted in the anamnesis of a patient with dysphagia: anxiety, insomnia, palpitations, burning hands, etc., guiding the semiotician to the diagnosis of functional neuromotor dysphagia, secondary to a neurovegetative, endocrine or psychic condition.

d) Sialorrhea, whose symptom associated with low dysphagia suggests cancer of the lower portions of the esophagus (Roger's sign).

e) In other cases, dysphagia is accompanied by general symptoms, weight loss, loss of strength, all of which guides the diagnosis of dysphagia due to malignant processes.

f) Fever, is characteristic of mediastinal abscesses, perforated diverticula, etc.

Course

In this last section of the clinical studies of the dysphagia symptom, we will summarize the fundamental characteristics that distinguish organic dysphagia as a whole from functional dysphagia.

The course of organic dysphagia is uniform, regular, rhythmic, progressive, with initial difficulty in swallowing solids, until ending in not being able to swallow even liquids. As an example, carcinoma of the lower portions of the esophagus is always pointed out, in which disease the patient's progressive cachexia, as well as hyperalorrhoea (Roger's sign), complete the clinical typicality of the diagnosis.

nostic.

The course of functional dysphagia is capricious, sometimes the dysphagia disappears in 1-2 days to reestablish itself later, irregular, arrhythmic, oscillating, with paradoxical difficulty swallowing liquids. and yet be able to swallow solids easily, sometimes giving in to atropine and cortical sedatives. The relative good general condition of the patients is notable, and they also report multiple psychovegetative symptoms.

SEMIODIAGNOSIS

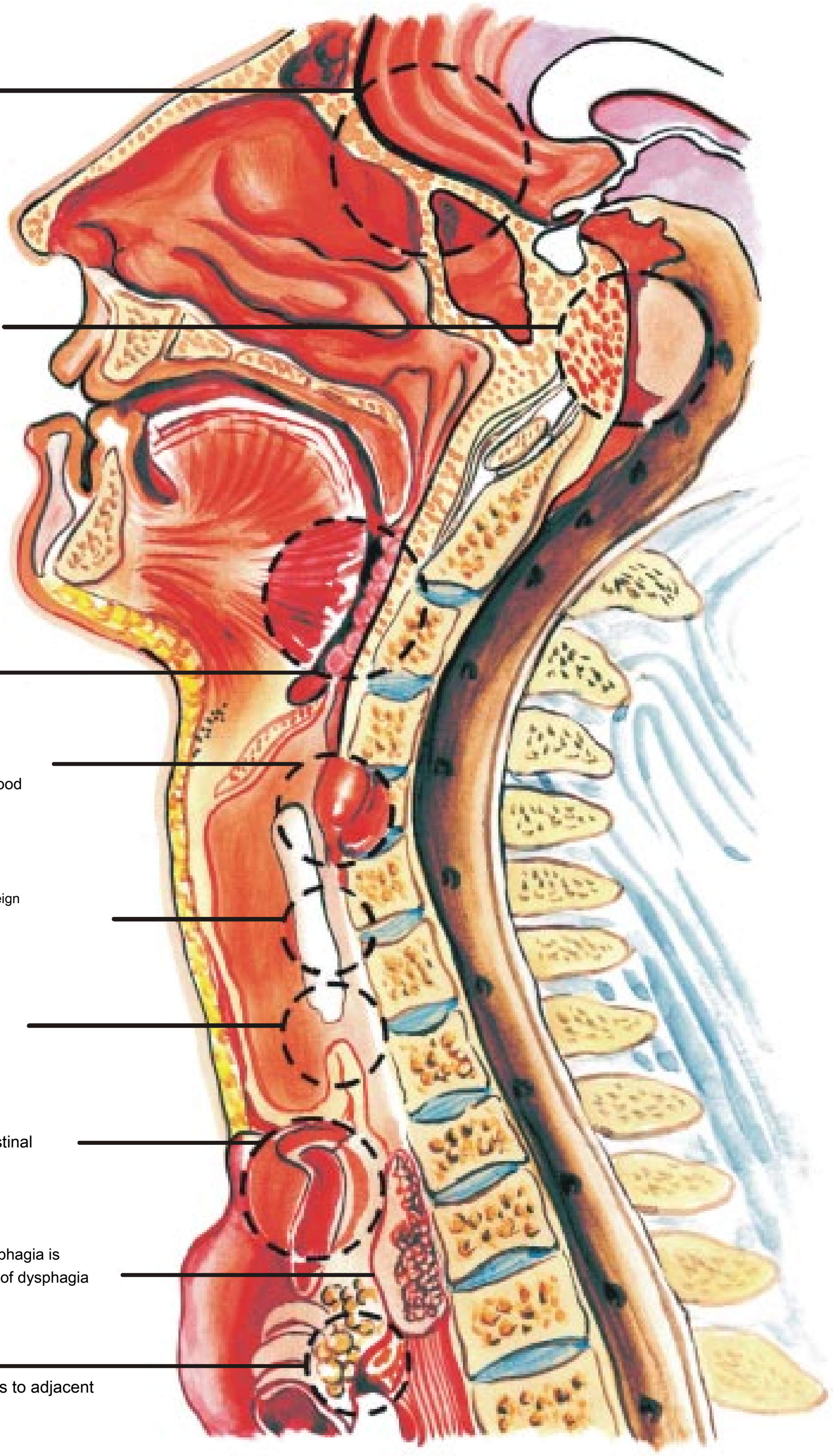
From what has been studied, it is clear that noting the dysphagia symptom in the anamnesis, although it should selectively guide us to think about diseases of the esophagus, also forces us to keep in mind that it can correspond to diseases outside the esophagus (of other systems) and even to diseases of a humoral nature (deficiency, endocrine, psychovegetative, allergic, etc.).

1. Organic dysphagia. When faced with organic dysphagia (fig. 56.4), we will think about the presence of benign tumors and more commonly malignant ones (carcinoma), located at any esophageal level. Also considered are strictures caused by chronic stenosing esophagitis, especially caustic esophageal stenosis in children due to accidental ingestion of potash; diverticula, foreign bodies, etc., are typical examples of intrinsic obstruction.

   Stenosis of extrinsic cause is due to the increase in size of the organs neighboring the esophagus, such as the heart, aorta, lung and other structures of the mediastinum (nodes, etc.).

2. Functional dysphagia. Let us point out as the most frequent diseases, pharyngitis, laryngitis, small diverticula and acute and subacute esophagitis, which acting as irritating thorns reflexively produce functional dysphagia. Lesions of the bulb and myasthenia gravis produce functional dysphagia due to cancellation of the swallowing reflex.

3. We can also mention, among the diseases that cause functional dysphagia, the essential types



PSYCHOGENIC DISORDERS.

Globus hysteriques, cardiospasm, and Plummer-Vinson syndrome may have a psychogenic component.

NEUROMUSCULAR DISORDERS.

Bulbar paralysis, bulbar poliomyelitis, myasthenia gravis, diphtheria polyneuritis.

OROPHARYNGEAL INFLAMMATION.

Pharyngitis, glossitis, tonsillitis, abscess, angioneurotic edema.

PHARINGOESOPHAGEAL DIVERTICULUM.

The associated symptom is regurgitation; Food seems to stay in the mouth.

BENIGN Narrowness.

Caused by ingestion of a caustic substance or foreign body; It also occurs in scleroderma.

ESOPHAGGOTRACHEAL FISTULA.

Mainly due to congenital defect,
It may also be due to a malignant tumor.

MEDIASTINAL TUMOR.

Aneurysm of the arch of the aorta, mediastinal tumor, diaphragmatic hernia.

NEOPLASIA.

The most common cause of obstructive dysphagia is carcinoma; Benign tumors are also a cause of dysphagia due to obstruction.

TRACTION DIVERTICULUM.

Caused by the adhesion of the esophagus to adjacent organs or formations; It is best verified by fluoroscopy.

Fig. 56.4 Organic dysphagia.

known by the names: diffuse spasm of the esophagus, transient spasm of the cardia and especially Hurst's achalasia. Finally, let us point out certain psychoneurotic and psychotic diseases as possible causes of functional dysphagia. Within the deficiency pathology, hypovitaminosis B and the well-known Plumme-Vinson syndrome are noted, in which iron deficiency plays an important role.

PYROSIS

CONCEPT

Let us define this symptom as the sensation of heat, burning or burning that the patient experiences behind the sternum, particularly in the vicinity of the third portion or xifisternum. This sensation tends to ascend behind the sternum to the pharynx, sometimes reaching a painful tearing character. The subjective sensations that make up the heartburn symptom are very variable. Alvarez, carefully noting the characteristics of this symptom, described by 123 different patients, was unable to fit them into a single identification pattern. It appears and disappears, sometimes, without a precise cause, evolving for more or less long periods.

SEMIOGENESIS 0 PATHOPHYSIOLOGY

We have already pointed out that heartburn is a mainly functional symptom. Let us analyze its intimate pathophysiological mechanism and the various causal

Intimate mechanism

Jones definitively proves the sudden distension of the esophagus as the cause of heartburn (in its intimate mechanism), by reproducing this symptom experimentally in volunteer human subjects. This author distended the supracardial esophagus with a balloon introduced through the mouth using a fine probe in these individuals, reproducing a high percentage of the sensation of heartburn. In turn, he observed that the physicochemical characters of some acidic or alkaline, hot or cold liquids did not seem to have an important role; On the contrary, the introduction of large quantities at high speed and pressure easily reproduced heartburn, coinciding with a manifest alteration of peristalsis, due to reversion to that level.

This phenomenon, clearly related to the quantity and pressure factor, was verified by Jones using a nasal probe, through which he instilled the different solutions of cold or hot water, N/10 HCI, N/10 NaOH, the subject's own gastric juice and barium sulfate. At the same time, he took advantage of fluoroscopic control and observed that the moment in which the subject experienced maximum heartburn coincided under the screen, with the arrest of the barium in the esophagus, and that nothing passed to the stomach through the cardia. On the contrary, in some cases retroperistalsis was observed towards the upper end of the esophagus. When the peristaltic arrest wave disappeared, the cessation of the heartburn symptom was observed. It is assumed that when an alkaline medication calms heartburn, it does so not by neutralizing the acid, but by regulating or stopping retroperistalsis. Bockus points out that the motor alteration is reminiscent of what occurs in esophageal pain itself, and that there is no difference other than the lower intensity of the reflex stimulus in the heartburn symptom.

Heartburn can also occur in cases of gastroesophageal reflux, mucosal irritation, or both.

SEMIODIAGNOSIS

There may be multiple diseases capable of producing heartburn, and essential or idiopathic heartburn is also acceptable. Gordon points out that in 500 dyspeptics reviewed, heartburn occurred in 82 9%, which is equivalent to 410 patients, of which it was considered as the main symptom in 46% and as an associated symptom in 54%.
Among these diseases we mention: heartburn of the hiatus hernia, with terminal esophagitis; as well as other lower esophageal and juxtadiaphragmatic injuries.

Let us also point out some types of chronic gastritis and, in general, many types of esophagogastric dyskinesias reflex to more or less distant lesions:
appendicitis, abdominal inflammatory processes, etc. Also important is heartburn in psychoneurotics, tachyphagic people and subjects who become upset while eating, and heartburn in allergic people, whether due to esophageal contact or endogenous cause.

Let us particularly note due to its frequency (60%), importance and resistance to treatment, heartburn in pregnant women; We have had the opportunity to verify the presence of this symptom in many of them throughout the pregnancy and its intensification in the last weeks, only to disappear with delivery. In this case, the pathogenic convergence of different factors seems evident: hormonal, vagosympathetic, psychological and sensitization or allergy alterations.

PITUITA

CONCEPT

This symptom is defined as the expulsion through the mouth, as vomiting, of the abnormal contents previously accumulated in the lower portions of the esophagus (esophageal regurgitation). It is often carried out on an empty stomach, which is why this symptom is also known as morning pituitary. Its synonymy is broader, with equivalent terms also cited as: pitituous vomiting (Mathieu), false vomiting and esophageal vomiting. The pituitary is identified with the quiet expulsion of a liquid that is generally clear, viscous or thin and not very abundant (60-120 mL).

SEMIOGENESIS OR PHYSIOPATHOLOGY

The expulsion of the material in the pituitary is done at the expense of the intrinsic musculature of the esophagus, possibly by retroperistalsis as an internal mechanism. This material accumulates previously in the lower portions of the esophagus for three fundamental reasons:

1. Salivary hypersecretion (sialorrhea) of any cause, saliva slips from the mouth to the lower esophagus.
2. Production of abnormal elements of the esophagus itself (in situ) in cases of diverticula, esophagitis, tumors, etc., generally they are mucus, bloody gums, food remains from a jam, etc.
3. Gastric reflux of variable acidity, whose secretion passes through the cardia and accumulates in the esophagus.

The accumulation of material in the esophagus can last for a longer or shorter time before pitiful vomiting occurs.

In cases of chronic alcoholics, for example, the accumulation of saliva and gastric secretion due to reflux occurs during the night while the subject sleeps, and is expelled upon awakening, hence the name morning pituitary for this modality.

SEMIODIAGNOSIS

1. Among the pituitary-producing diseases, due to sialorrhea and sialophagia, we mention the pituitary of smokers and alcoholics.
2. When faced with a pituitary gland, we must also think about esophagopathies such as: lower diverticulosis, some types of esophagitis and esophageal achalasia.
3. Finally, let us point out chronic gastritis as a cause of pituitary.

DIGESTIVE SYSTEM. SYMPTOMS OF THE GASTRODUODENOHEPATOBILIOPANCREATIC

57

Introduction

The symptoms corresponding to this segment can be grouped into:

- Great symptoms or capital symptoms
  - abdominal pain
  - Vomit
  - Hematemesis
- Small symptoms or dyskinetic or dyspeptic symptoms
  - Aerophagia or aerophagic belching
  - gastric regurgitation
  - Non-aerophagic belching
  - Gastric burning
  - Hiccup
  - Gastric fullness Heaviness and satiety Nausea
  - epigastric jump
  - Other vague subjective symptoms, sensation of pressure, emptiness, etc.

This is a very extensive and important group of subjective symptoms of the digestive system. The reference to the stomach, duodenum and hepatopancreatic system is not absolute, since in many circumstances these symptoms may correspond to other segments of the digestive system, as well as to processes located outside its limits, but without a doubt in the presence of said symptoms we should preferably think about such a reference.

Due to their evident range, we have divided these symptoms into two large groups, since when faced with epigastric pain, vomiting or hematemesis, we think, not without reason, of processes of greater importance, and not so when faced with the presence of the so-called dyskinetic or dyspeptic symptoms. The first, large symptoms or capital symptoms, most frequently depend on processes of an organic nature, and the second, small symptoms, are generally typical of functional processes. This rule is not absolute, but it is a very useful guide.

The capital symptoms or major symptoms - abdominal pain, vomiting and hematemesis - in turn take on great semiological value if they occur simultaneously in the anamnesis of a patient with digestive disorders.

The simultaneous presence in a patient of these three major symptoms is known as the great digestive triad, which very often corresponds to an organic lesion of the stomach or duodenum. The great symptomatic triad of gastric conditions, cited by the French school, strongly guides the diagnosis of an ulcer, cancer or other advanced organic lesion of the stomach, with participation of the sphincter orifice of the pylorus. The presence in the patient of only two of these three symptoms, although it reduces the indicated value, continues to point towards that possibility. The presence of just one of these capital symptoms requires further semiographic analysis to be able to classify it in a particular organ.

As we said before, the symptoms of the second group, called dyspeptic symptoms, are also called dyskinetic symptoms. This term dyskinetic is very correct, as we already explained in the previous chapters dedicated to the digestive system, particularly in that of general pathophysiology. These symptoms, of a motor nature, are mostly due to functional alterations that depend on the disturbance of gastroduodenal motility, tonus, or peristalsis.

These dyskinetic phenomena can be due to multiple causes that may lie in the upper digestive segment itself or at a greater distance from it.

In the synoptic table that begins this chapter, we have made a general listing of these small symptoms, without making any definitive subclassification of them. It is appropriate that we say here that in the more or less classic works of digestive semiology these dyspeptic or dyskinetic symptoms are usually classified as hypersthenic and hyposthenic. Those symptoms are designated as hypersthenic for which, in some way, in their mechanism of production or semiogenesis, a certain increase or paradoxical irritability of the altered gastric functions is assumed, and those others that have been considered as resulting from a pathological decrease or attenuation of the functions of the stomach are classified as hyposthenic.

Although this is verified to a certain degree, in many cases it does not always occur, and the same symptom can be found, say the presence of nausea, gastric fullness or another of these symptoms, linked indistinctly to phenomena of gastric hyperesthenia or hyposthenia. Chemical hyperchlorhydria (examination of gastric juice) is sometimes found in subjects with hyposthenic dyspeptic symptoms and the opposite, that is, hypochlorhydria in hypersthenic dyspeptics.

Other symptoms of this group are so imprecise and particular in their semiogenesis that no subclassification can be attempted about them. Nausea as an isolated symptom (not followed by vomiting), epigastric jump, as well as the sensation of pressure, emptiness, etc., are imprecise symptoms that we have also included in this group of small symptoms, to try to study them in the best possible way.

Let us also repeat here, as we said in previous chapters, that some of these symptoms of the gastric segment could be included within a neoanatomical or physiological classification, as corresponding to the juxtacardial segment. They are: aerophagia and gastric regurgitation, whose semigenetic relationships with low dysphagia, pituitary and heartburn (symptoms already studied in the oropharyngoesophageal segment) are very evident, all expressing together the neurogenic alteration of the supracardial and infracardial area.

BIG SYMPTOMS0 CAPITAL SYMPTOMS

The major symptoms or capital symptoms are:

- Abdominal pain.
- Vomiting.
- Hematemesis.

ABDOMINAL PAIN

We have considered the convenience of unifying epigastric pain and non-epigastric pain in a single section with the term abdominal pain, since their general pathophysiology is common.

Preliminary considerations

The organism, despite its unity, presents a profound metamerism, so it can be considered divided into a series of segments, each one derived from a pair of embryonic somites. Each somite, as it evolves, differentiates into three portions (Hamilton; Boyd):
an external one or dermatome, which will give rise to the dermal chorion and from which the skin from the ectoderm will receive its innervation; another internal one or myotome, from which the skeletal muscles come, and another medial or sclerotoma, origin of the bones, cartilage and other structures of the skeleton. The dermatone, the myotome and the sclerotome from the same somite receive sensory innervation from the same posterior nerve root from the corresponding neurological segment. The myotome also receives a motor root.

Torah.

The segmental distribution of dermatomes is best observed on the thorax and abdomen, where they are distributed in narrow circular bands around the trunk. However, also in the arms, if they are kept horizontal, it is observed that the dermatomes run parallel from the inside out, and the same occurs in the lower extremities, even though in these the distribution is masked by the subsequent rotation they undergo during embryonic development, in such a way that they follow a spiral course around the limb, from top to bottom and from outside to inside on the anterior surface, and from inside to outside on the posterior surface.

The viscera do not develop from the somites. However, its sensory innervation is carried out, as in somatic structures, by fibers from the posterior roots, although they reach it through the sympathetic white rami communicantes, for which reason the viscera also show, through their innervation, a certain segmental distribution.

Concept

We could define abdominal pain as the subjective manifestation, always unpleasant, more or less intense, ranging from just a painful sensation to sometimes something desperate, perceived by the patient anywhere in the abdomen.

Semiogenesis or pathophysiology

Like all pain, abdominal pain is a symptom of a neurogenic nature that requires the presence of:

1. An irritating thorn or stimulating factor.
2. A local receiving system.
3. Nervous pathways to reach the central receptor areas (thalamus, post-Rolandic cerebral cortex).

The pathogenic theories of epigastric abdominal pain are multiple and inaccurate, and before analyzing them it seems convenient to remember the concepts referring to the metameric zones.

Let us remember the data of applied anatomy and physiology, essential to understand the pathophysiology.

The stomach, duodenum and other viscera of the upper abdomen are innervated by the vagus or pneumogastric nerve, which represents the parasympathetic system, and by the splanchnic nerves, which carry fibers from the great sympathetic nerve, particularly the greater splanchnic nerve, which corresponds to the D5-D9 spinal cord segment and ends in the semilunar ganglion. From this ganglion, the fibers pass to the solar plexus, the center of sympathetic innervation and the site where the synapse is made. In the gastric viscus, therefore Therefore, there is the aforementioned double vagosympathetic innervation, in intimate relationship with the gastric plexuses themselves (Auerbach, Meissner, etc.).

This innervation constitutes the efferent pathway (from the center to the periphery) made up of preganglionic efferent elements that, coming from the medulla, cross the anterior root and the white communicating rami, to reach the ganglion, and postganglionic efferent elements that go from said autonomic ganglia to the interior of the stomach. In turn, let us also mention the pneumogastric route as an efferent route.

The afferent pathway (from the periphery to the center) starts from the gastric viscus and crosses the autonomic ganglia at the level of the plexuses, but without making contact synapses with their neurons. This afferent pathway, through the splanchnic nerve, reaches the spinal cord, in association with the somatic afferent fibers (central nervous system) at the level of the same nerves. Both visceral (from the stomach) and somatic (from the parietal areas of the upper abdomen) nerve fibers establish reflex connections with the neurons of the autonomic system in the spinal cord.

The afferent pathway, as we said, crosses the autonomic ganglion from the viscus without connecting, it also crosses the sympathetic chain without any connection; It continues along the white rami communicans, travels with the spinal nerve in the direction of the medulla, and it is at the level of the dorsal spinal ganglion where it synapses with the dorsal sensory root of the central nervous system. There it meets with the fibers of the peripheral sensory nerves, then follows a common path to the centers (through the medulla), reaches the nuclei of Goll and Burdach (where it synapses) and continues along Reil's ribbon to the thalamus. (true center of pain). When the intensity of the stimulus is greater, it reaches the cerebral cortex, making the painful sensation conscious (fig. 57.1).

The response impulses go towards the periphery following the efferent pathways already described. Let us also mention, on the other hand, the vagal or parasympathetic afferent route from the stomach, which is of much less importance.

The anatomical review that we have done, which demonstrates the intimate connection of the autonomic system, particularly the sympathetic pathway with the cerebromedullary nervous system, is essential to understand Mackenzie's viscerosensory reflex, one of the most important pathogenic theories of epigastric pain.

Likewise, we have remembered through anatomy the direct, somatic connections of the visceral peritoneum with the central nervous system, data that will also be used when we explain the pathogenic theories of epigastric pain.

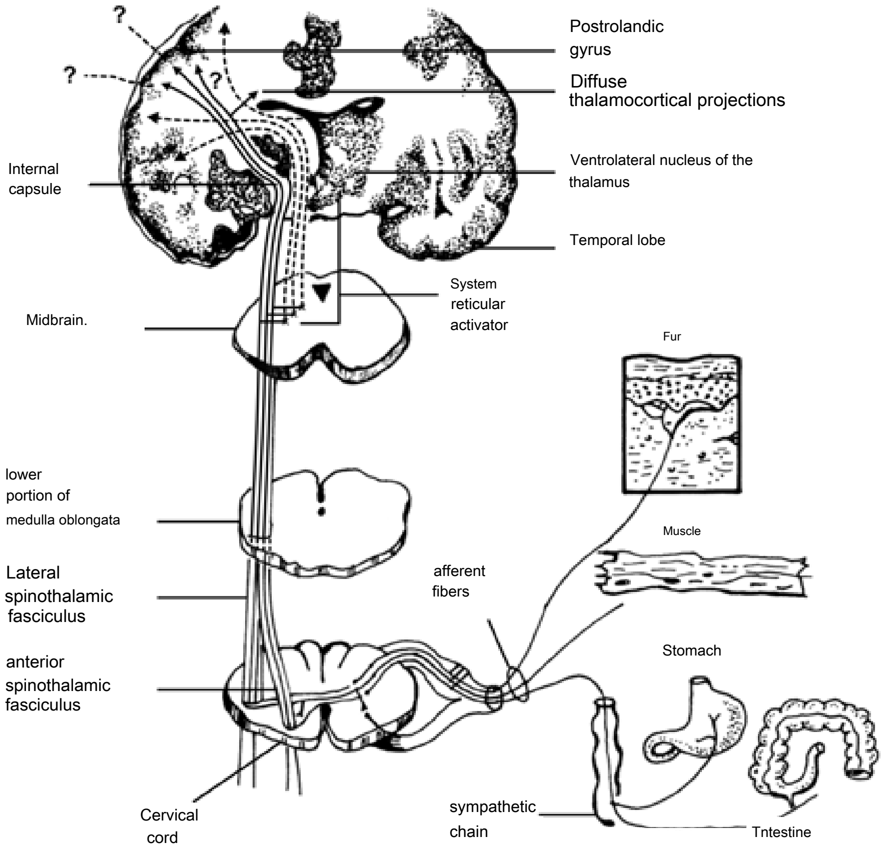

Postrolandic gyrus
Diffuse thalamocortical projections
Ventrolateral nucleus of the thalamus
Temporal lobe
Internal capsule
System reticular activator
Midbrain.
Fur
lower portion of medulla oblongata
Muscle
Lateral spinothalamic fasciculus
afferent fibers
Stomach
anterior spinothalamic fasciculus
Cervical cord
sympathetic chain
Tntestine
?
?
?

Fig. 57.1 Pathways of somatic or referred pain and visceral or true pain.

Theory of the viscerosensitive reflex, by Ross and Mackenzie

This theory considers that the afferent pathway of the pain reflex begins in the viscera, is transmitted through the great sympathetic fibers included in the splanchnic nerves (especially the greater splanchnic nerve for epigastric pain), crosses the corresponding sympathetic ganglion, travels through the white rami communicans, through the corresponding spinal nerve and reaches the posterior root ganglion where it has its neuron (as seen it is a long dendrite). The cylinder axis of this neuron travels through the posterior root and upon entering the spinal cord it establishes synapses in the lateral and anterior horns.

At this level, a focus of irritation occurs and at the expense of the connection that is made with the nerves of the relationship life, posterior roots and peripheral nerves, a phenomenon of nervous induction or deviation is established at that level. On the one hand, these afferent impulses follow the ascending pathways to reach the receptor centers of the brain (spinothalamocortical pathway), and on the other hand, these autonomic afferent impulses travel along the peripheral nerve pathway, and the painful sensation (referred pain) is located in the somatic or parietal area corresponding to the own metameric level of the zone of spinal irritation, at a greater or lesser distance (metameric deviation and association). This pain requires a strong stimulus, is acute and well defined, and is called epicritic (Fig. 57.2).

Theory of pure visceral pain

The theory of pure visceral pain (Leriche, Ryle and others) has little clear evidence. Let us mention, however, how White, Garrey and Atkins, among others, demonstrate the production of the pain reflex, even if the posterior roots of the spinal cord are sectioned: that is, pure viscerosympathetic pain. These authors prove that pain only disappears if the splanchnic or communicating ramni are sectioned, that is, the sympathetic pathway to the centers. The authors themselves, however, point out that when cutting the peripheral pathways or blocking the wall with novocaine (Weiss and Davis), the pain that persists (pure visceral) is different, that is, it is poor, dull, diffuse and deep (protopathic pain), in short, different from the so-called referred pain that occurs when the connection with the periphery or abdominal wall is maintained. This reflex requires adequate and intense stimuli that come from

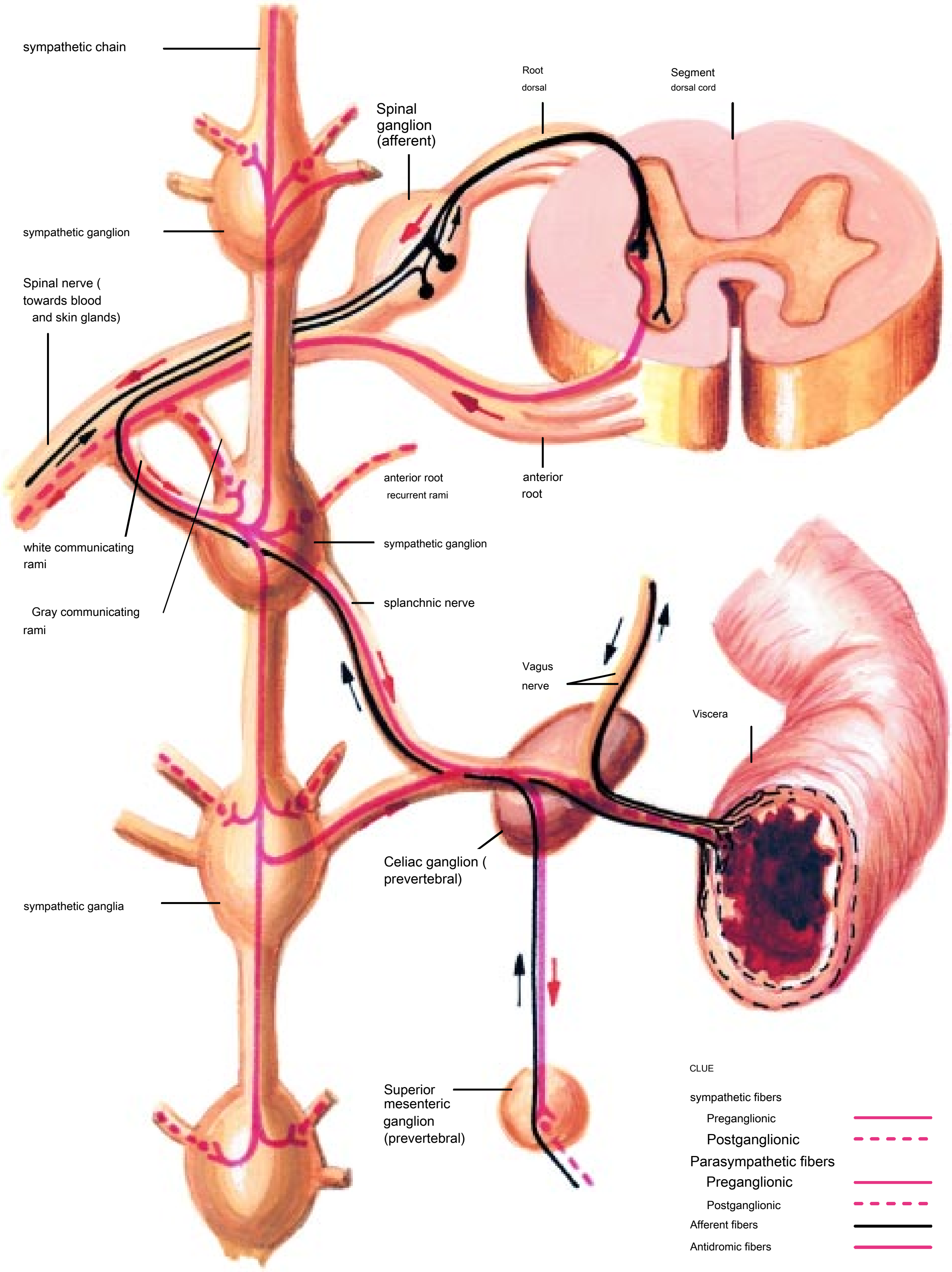

sympathetic chain
Root
dorsal
Segment
dorsal cord
Spinal
ganglion
(afferent)
sympathetic ganglion
Spinal nerve (
towards blood
and skin glands)
anterior root
recurrent rami
anterior
root
white communicating
rami
sympathetic ganglion
splanchnic nerve
Gray communicating
rami
Vagus
nerve
Viscera
Celiac ganglion (
prevertebral)
sympathetic ganglia
Superior
mesenteric
ganglion
(prevertebral)
CLUE
sympathetic fibers
Preganglionic
Postganglionic
Parasympathetic fibers
Preganglionic
Postganglionic
Afferent fibers
Antidromic fibers

Fig. 57.2 Viscerosensory reflex.

of its own environment: distension produced by trying to overcome resistance, spasms, chemical irritations and mechanical actions exerted on the hyperemic organ.

Morley's Peritoneosensory Reflex Theory

Morley's theory tries to deny the pathogenic conceptions described above. It combats the existence of both referred or visceral pain and pure visceral pain. Morley denies the intervention of the afferent autonomic pathways indicated by Mackenzie and says that referred pain originates only from irritation of the somatic nerves of the parietal peritoneum or mesentery, and that the painful sensation refers to the area of the same segment. For this author, this explains why the peritoneum or mesos are sensitive to different stimuli while the viscera itself is not. For Morley, pain is a peritoneosensitive reflex.

Theory of vagal reflex transmission

Conduction to the centers of painful reflexes from the stomach or a diseased viscus through the vagal or pneumogastric route has also been reported, but it seems that this is carried out with much less possibility (Cannon).

Intimate mechanism or visceral onset of the pain reflex

We already pointed out when talking about the pure visceral or sympathetic reflex, the need for an adequate stimulus generated in the viscera itself. Hurst, from the English school, demonstrated with his experiments that there is a phenomenon of visceral tension (distension) as a primary factor in the origin of pain. It is not purely the phenomenon of contracture that generates the pain reflex, but rather the inability to relax or achalasia, or spastic obstruction of the neighboring sphincter, seems to be necessary. These motor alterations are similar to those mentioned regarding the pathophysiology of low dysphagia and heartburn.

These initial motor phenomena of the pain reflex can be considered always present to a greater or lesser degree and common to any of the theories of pain propagation that we have previously explained, although they are particularly indispensable as an adequate stimulus, when the theory of pure viscerosympathetic pain is explained.

Eclectic pathogenic theory

In some cases it is acceptable that from the primary visceral motor phenomenon, pure visceral pain is only established if there is a discrete neuronal irritation at the level of the spinal ganglion with the intervention of the sympathetic fibers only, producing (protopathic) pain: functional, dull, diffuse, poor, etc. In other circumstances, based on the motor phenomenon itself visceral, more intense afferent impulses will be added, to such a degree that, in addition to propagation through the sympathetic pathway, they are capable of creating a much more marked focus of spinal irritation, exciting the secondary neurons of the spinal cord and reflecting to the superficial planes, producing referred pain (epicritic): acute, intense, more localized, etc. with sensory and motor equivalents in the corresponding metamer or in other associated metamers.

From the common primary visceral motor component, somatic pain can be determined by extension of the visceral neuronic lesion and by contact with the peritoneum or mesentery, similar to any pain in the life of a relationship: organic and migratory pain with the movements of the viscera, of an externalizing nature as proposed by Morley.

Let us also point out the role that the vessels play in the initiation of the pain reflex. In cases of spasms or inflammation of the arterial wall, as Bockus cites, a painful, intense, growing, sometimes agonizing reflex is established, as occurs in many cases of hepatic colic, which reminds us of the mechanism of anginal pain due to coronary alteration. Something similar happens when the pain comes from a narrow duct such as the common bile duct. This pathophysiological knowledge will be very useful when we study pain semiography, especially in relation to quality and intensity.

Later, starting with Vanei's work in 1970, other horizons were opened that tended to delve deeper into the intimate mechanism of pain. This author demonstrated that certain non-steroidal anti-inflammatory substances, such as aspirin and indomethacin, inhibit the synthesis of some prostaglandins and their metabolites. This suggests that prostaglandin could act by increasing the sensitivity of nerve receptors and thus facilitate or produce the onset of this symptom.

In summary, the three types of pain must be accepted: pure visceral pain (viscerosympathetic reflex), referred pain (viscerosensitive or sympathomedullary reflex) and pure somatic pain (peritoneosensory or cerebrospinal reflex); The attention of the semiotician is drawn, that of the three it is Mackenzie's referred pain with its direct or associated metameric references, which is the most interesting, as we will see later, from the semiographic, characterological point of view or the assessment of epigastric pain in the daily clinic.

Finally, let us point out other accessory factors that also influence the pathogenesis of epigastric pain and which are: the abdominal wall, the pain threshold and the psychosomatic factor, as well as the existence of silent visceral areas. All of them are important in the clinical assessment.

In general terms, the diseases that the semiotician must think about, when faced with the existence of subjective epigastric pain, are extraordinarily numerous. We must repeat, however, that according to the law of the distance of the irritating spines and the conception of the effector organ, it is more logical that when faced with epigastralgia we think preferably of a gastroduodenal disease or of a neighboring organ of the upper abdomen: liver, bile ducts, pancreas, etc., but diseases of the rest of the digestive system should also pass through our mind: jejunum, fleon, colon, peritoneum, mesos, appendix, etc.

At the same time, we will keep in mind the extradigestive diseases of the abdominal wall itself, respiratory, circulatory, renal, hematopoietic organs, the endocrine system, nutrition, the neurovegetative system, the central nervous system, the spine and the diseases of the psyche (psychopathies and psychoneuroses).

The semiogenesis of non-epigastric abdominal pain is similar to that mentioned above for epigastric pain.
However, we must remember, in the order of clinical anatomy and physiology, that parasympathetic innervation is carried out in the intestine through the pneumogastric to the middle of the transverse colon, and from this level to the rectum inclusive, through the pelvic or erector nerve, which emerges from the second, third and fourth sacral segments. Sympathetic innervation is carried out in the intestine and in the lumbocolonic segments from the mesenteric plexuses, and at lower levels, from the hypogastric plexus, by the presacral nerve, establishing the same reflexes as at the higher levels through the rami communicantes (fig. 57.3).

The metameres corresponding to the intestine begin approximately at D9, include the rest of the dorsal cord and continue with the lumbar and

sacral

The lesions of the intestine acting as irritating thorns are, therefore, at lower levels than those cited for epigastric pain, but the reflexogenic phenomena are the same, the same viscerosensitive, pure visceral, and peritoneosensitive reflexes are produced from the visceral motor alteration itself, as occurs in the production of epigastric pain. In the pathophysiology of non-epigastric abdominal pain, finally, accessory factors also play a role: parietal reflex, pain threshold, silent areas and psychosomatic factor.

Semiography and semidiagnosis

The simple note, in the interrogation of a digestive patient, of abdominal pain or epigastric pain will guide little or nothing in the diagnosis; However, the careful notation of its characters is sometimes so fortunate that it allows us to practically make a diagnosis, which can be confirmed by other complementary resources, as happens, for example, in many cases of duodenal ulcer or calculous cholecystitis.

This concept requires studying in detail the semiography of the fraud, its characterology or clinical study, which is summarized in 11 semiographic characters, listed in table 57.1.

Table 57.1

Semiographic characteristics of abdominal pain
1. Location 2. Irradiation 3. Periodicity 4. Rhythm or schedule 5. Intensity 6. Quality or character 7. Mode of beginning 8. Way to calm down 9. Variation according to position changes 10. Associated symptoms 11. Course

The most essential factors to analyze within this study are:

1. Location. In relation to the pain located in the epigastric region, we must understand as such, that which the patient refers, from the xiphoid to the navel and more or less central (Labbe's triangle and Mendel's painful zone, from the anatomists). It is very useful for diagnosis to consider, as patients often do, high, medium, low epigastric pain, as well as locations further to the right or further to the left. As an exception, without feeling anterior or epigastric pain itself, sometimes patients report posterior pain, at the level of the thoracolumbar column, more or less corresponding to the level of the height of the epigastrium. These location details must be analyzed due to their high semiological value.

To analyze the location of abdominal pain located in the other regions of the abdomen that are not the epigastrium, we must remember the anatomical projection of each region (fig. 57.4) and consider the metamers corresponding to the lower jejunum, ileum, colon and rectum, with the different reflexogenic levels. The different segments of the small and large intestine have the following metameric levels of painful projection (as verified by Jones when suddenly distending the intestine at different levels, by introducing balloons through the mouth or anus) (figs. 57.5, 57.6 and 57. 7).

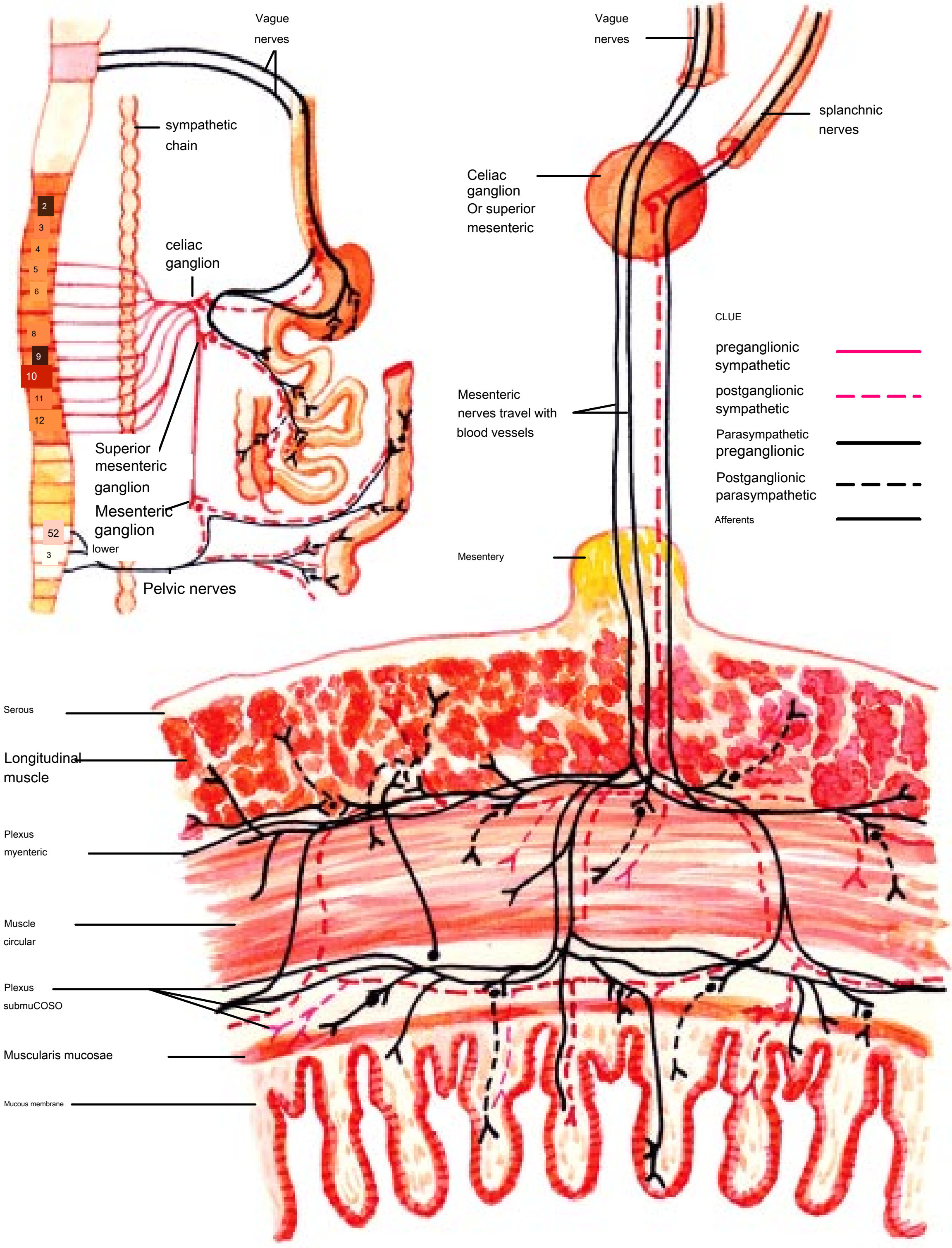

Vague nerves
sympathetic chain
2
3
4
5
6
8
9
10
11
12
celiac ganglion
Superior mesenteric ganglion
Mesenteric ganglion
52
3
lower
Pelvic nerves
Vague nerves
splanchnic nerves
Celiac ganglion Or superior mesenteric
Mesenteric nerves travel with blood vessels
CLUE
preganglionic sympathetic
postganglionic sympathetic
Parasympathetic preganglionic
Postganglionic parasympathetic
Afferents
Mesentery
Serous
Longitudinal muscle
Plexus myenteric
Muscle circular
Plexus submuCOSO
Muscularis mucosae
Mucous membrane

Fig. 57.3 Innervation of the intestine.

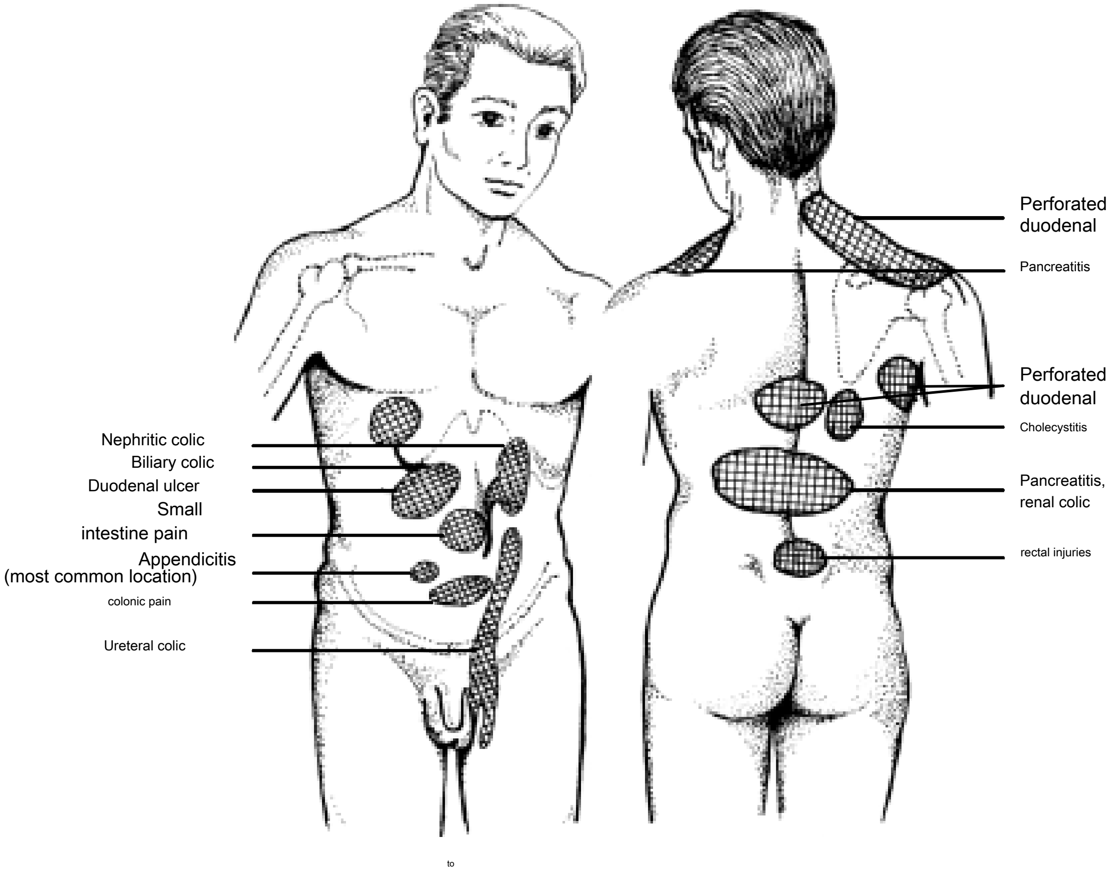

Nephritic colic
Biliary colic
Duodenal ulcer
Small
intestine pain
Appendicitis
(most common location)
colonic pain
Ureteral colic
Perforated
duodenal
Pancreatitis
Perforated
duodenal
Cholecystitis
Pancreatitis,
renal colic
rectal injuries
to

Fig. 57.4 Most common areas of referred pain: a, anterior wall; b, posterior wall.

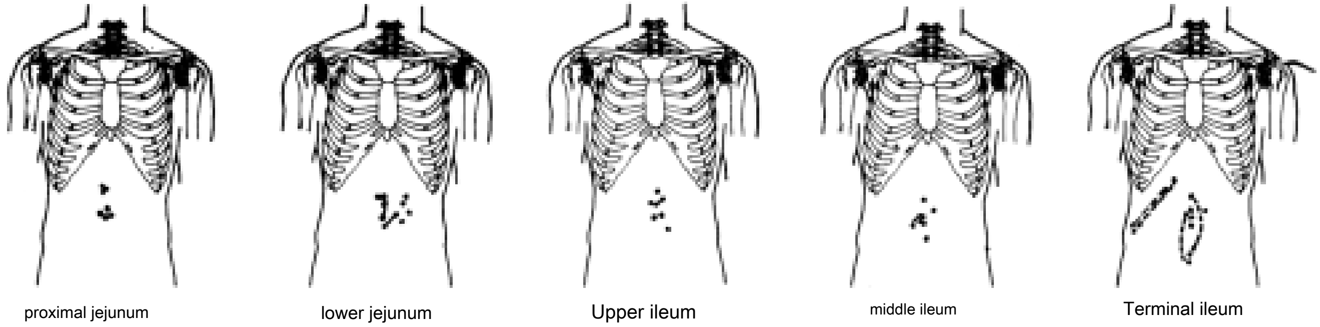

proximal jejunum lower jejunum Upper ileum middle ileum Terminal ileum

Fig. 57.5 Reference points of pain produced experimentally by distension of the jejunum and ileum.

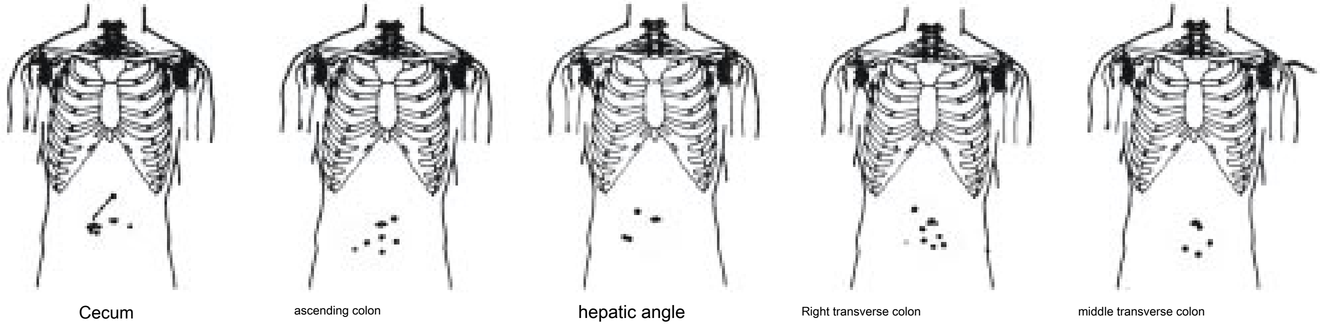

Cecum ascending colon hepatic angle Right transverse colon middle transverse colon

Fig. 57.6 Reference points of pain experimentally produced by distension of the right colon.

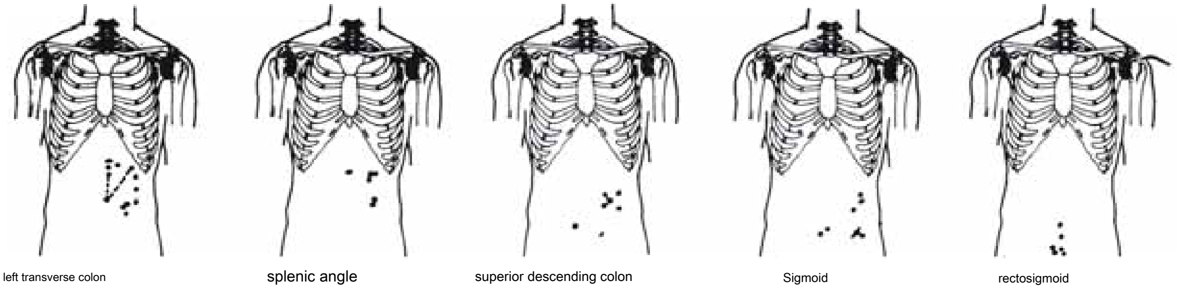


Fig. 57.7 Reference points of pain produced experimentally by distension of the left colon.

Jejunum	Behind: D12 to L5 Front: below the navel (maybe more to the right)
Cecum or terminal	Behind: L3 to L5 Front: right iliac fossa, navel and epigastrium
Colon ascending and epigastrium	Behind: L1 to L3 Front: navel, right and left
transverse colon-	Behind: L1 to L3 JFront: left hemiabdomen, midepigastrium to hypogastrium
Descending colon	Back: L1 to L5 Front: left hemiabdomen and hypogastrium
sigmoid colon	Behind: L3 to L5 Front: hypogastrium, left iliac fossa and perineum
Rectum and anus	Behind: up to the coccyx Front: lower hypogastrium and perineum

Regarding the location and its semidiagnosis, the pain can be:

a) High epigastric pain. This location should correspond to lower esophageal lesions, upper gastric lesions and, in general, functional alterations or juxtacardial dyskinesias, both intrinsic and extrinsic. Finally, we must also think about lesions of the coronary artery of the aorta.

b) Medium epigastric pain. It is observed in gastric lesions more or less adjacent to the pylorus and in hepatovesicular and pancreatic diseases. Other times, central and median epigastric pain is due to appendiceal processes (epigastric appendiceal reflex), extradigestational, particularly of a psychovegetative and neurological type (tabes, etc.), which produce this localization modality because it is the most frequent and nonspecific.

c) Low epigastric or superior juxtaumbilical pain. This modality suggests diseases of the distal portions of the duodenum and the duodenojejunal angle, as well as some alterations of the jejunum, ileum and particularly the ileocecal region, as Jones has been able to verify through experimental distensions with balloons.

d)Posterior pain (back). The semiotician must keep in mind the possibility that back pain is the only manifestation of a painful reflex (equivalent to the epigastric one), dependent on similar causes. Indeed, back pain at a variable level within the thoracolumbar segment (D7 to L2) may not be due to chest injuries, nor should it be considered an expression of a psychoneurosis, but rather be caused by diseases of the upper abdomen. For example, some types of juxtapyloric and duodenal bulb perforating ulcers in the pancreas, pancreatic injuries, diaphragmatic hiatus hernia, etc.

Berk, Chauffard and others have insisted on the semiological importance of posterior pain in pancreatic tumors (fig.57.8). Of course, the doctor must also think that the first thing is to rule out the multiple extra-digestive diseases that logically cause back pain, such as: spinal column disorders, trauma, tumors, systemic conditions, neurological conditions, etc., which go beyond the limits of digestive semiology.

e) Right epigastric and right hypochondrium pain. Pain located in the upper right quadrant of the abdomen (Anglo-Saxon topography) suggests preferably hepatobiliary diseases, angular colitis, as well as other

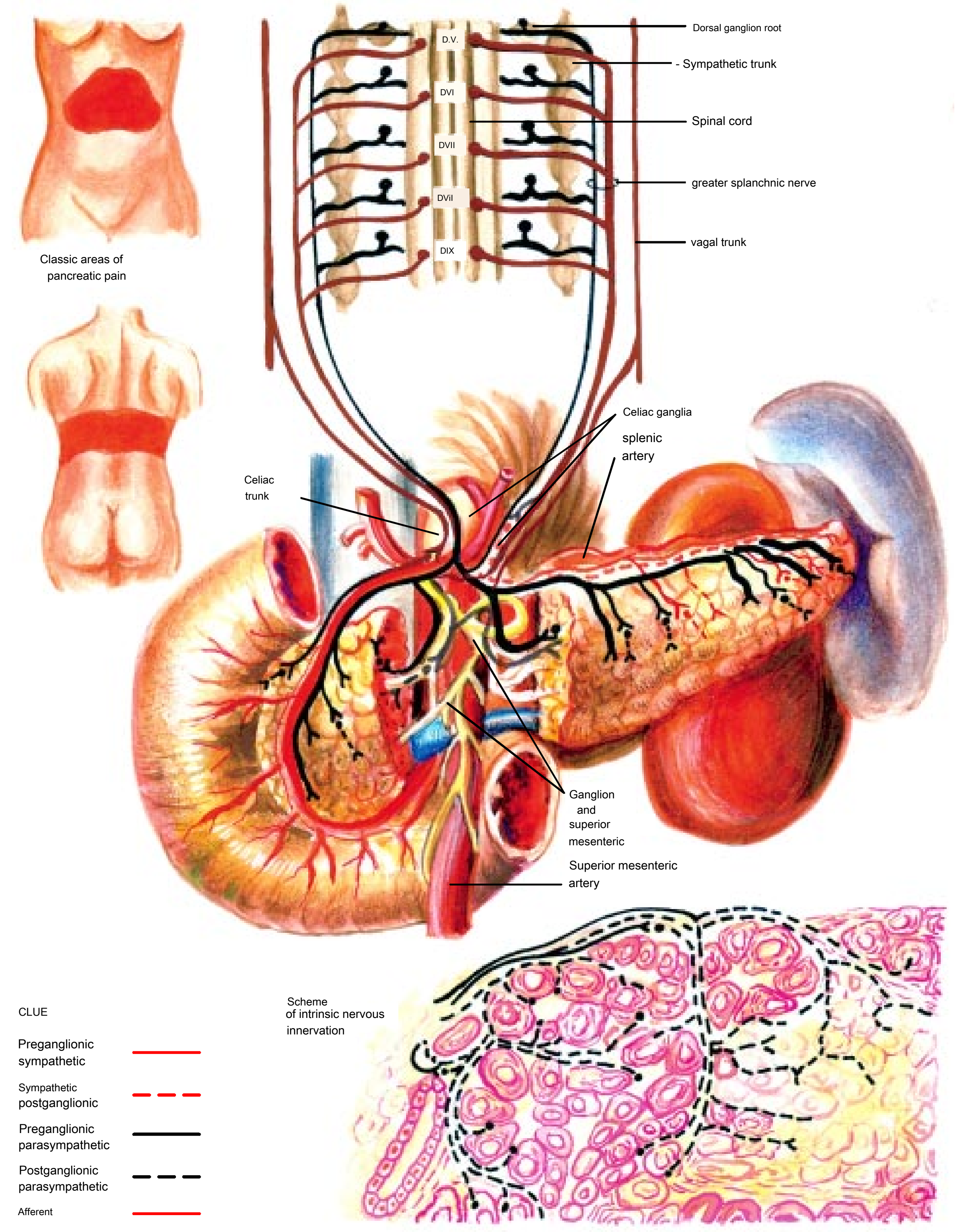

Classic areas of
pancreatic pain
D.V.
DVI
DVII
DVil
DIX
Dorsal ganglion root
- Sympathetic trunk
Spinal cord
greater splanchnic nerve
vagal trunk
Celiac ganglia
splenic
artery
Celiac
trunk
Ganglion
and
superior
mesenteric
Superior mesenteric
artery
Scheme
of intrinsic nervous
innervation
CLUE
Preganglionic
sympathetic
Sympathetic
postganglionic
Preganglionic
parasympathetic
Postganglionic
parasympathetic
Afferent

Fig. 57.8 Pain in pancreatic tumors.

parietal conditions at that level (neuritis, fibrositis, etc.), some nephropathies and some processes of the right pleura and diaphragm, among others. The aspect that we will analyze in greater detail will be hepatobiliary conditions.

We can sometimes distinguish three modalities of this type of hepatobiliary pain:

- Pure gallbladder pain. Located in the epigastrium, with a right orientation, it apparently corresponds to pure vesicular alteration; The pure visceral pathogenic reflex due to intravesicular hyperpressure intervenes in it. It occurs in lithiasic cholecystopathies and in non-lithiasic or dyskinetic cholecystopathies.

  Biliary pain (gallbladder, common bile duct, etc.). Epigastric and right hypochondrium location, with large irradiations that fundamentally follow the phrenic nerve. The patient with his gestures, as Gutmann points out, indicates to the doctor how the pain, leaving the epigastrium, goes to the right hypochondrium and the lumbar region, ascending from behind to the interscapulovertebral space and the anterior region of the chest, and often reaches the right shoulder and arm (fig. 57.9). Sometimes it spreads underneath, to the rest of the abdomen. A clear orientation to the left always suggests pancreatic participation.

- This type of pain, which is easily identified by the doctor and by many patients as hepatic colic or biliary colic (as it is said with greater pathogenic correctness), generally corresponds to gallstones or, with less probability, to processes of another nature, but involving not only the gallbladder, but the rest of the biliary tract, especially the common bile duct. In this pain, the mechanism of the viscerosensitive reflex of

  Mackenzie, with great bombardment, let's say with emphasis, through the sympathetic, on the focus of spinal irritation and even the Morley mechanism, accepting a peritoneal alteration with a somatic or cerebrospinal type reflex.

  Parenchymal liver pain. Location, only in the right hypochondrium; low amplitude, dull, generally produced by reflex from distention of Glisson's perihepatic capsule. It corresponds to liver diseases themselves: acute or chronic hepatitis, liver tumors, liver abscess: cardiac liver, etc.

f) Left epigastric pain. Among the diseases of the upper abdomen we must keep in mind as possible causes: postoperative peptic ulcers (pain more on the left) near the navel, a phenomenon explained by the anatomical projection of the anastomotic mouth; some hiatus hernias and injuries to the pancreas (body and tail). As we will see, due to non-epigastric abdominal pain, this type of pain can also be due to other diseases of the intestinal segment or diseases of the kidney, heart, left pleura and splenic conditions with perisplenitis.

g) Right hypochondrium. The pain in this region was partially studied when we referred to epigastric pain and parenchymal liver pain. However, it is necessary to emphasize that there are pains that are very limited to the right hypochondrium, corresponding to cholecystitis, pericholecystitis, empyema and the liver diseases themselves already mentioned, such as cardiac liver, liver neoplasms, hepatic steatosis, liver abscess, etc. We must also include some pancreatopathies with a particular location in the head, duodenitis, right juxtadiaphragmatic conditions and, finally, kidney pathologies should not be forgotten.

h) Left hypochondrium. The pain here suggests disorders of the spleen, left angular colitis, pancreatopathies, kidney and adrenal disorders on that side and the neighboring diaphragmatic area, and neuromuscular disorders in the region. Paradoxically, on occasions, biliary conditions can cause pain in this area.

i) Mesogastrium or umbilical region. It corresponds to diseases of the jejunum and ileum, from the duodenojejunal angle to the ileocecal valve: jejunitis and ileitis. Let us also point out some colonopathies of the transverse, abdominal aortitis, neuralgia of the mesenteric plexuses, as well as saturnine collocations and tabes. These latter processes can also cause pain in the upper abdomen, which covers this entire area in a belt.

j) Emptiness or left flank. The pain in this area corresponds to left or descending colitis and urinary pathology on that side: possibilities parallel to those mentioned for the right flank are pointed out.

k) Right iliac fossa. The pain is caused by appendicitis, typhlitis, ileitis, ileocecal tuberculosis, tumors of the ileum and cecum, perivisceritis, both congenital (Lane's band and Meckel's diverticulum) and acquired perivisceritis and

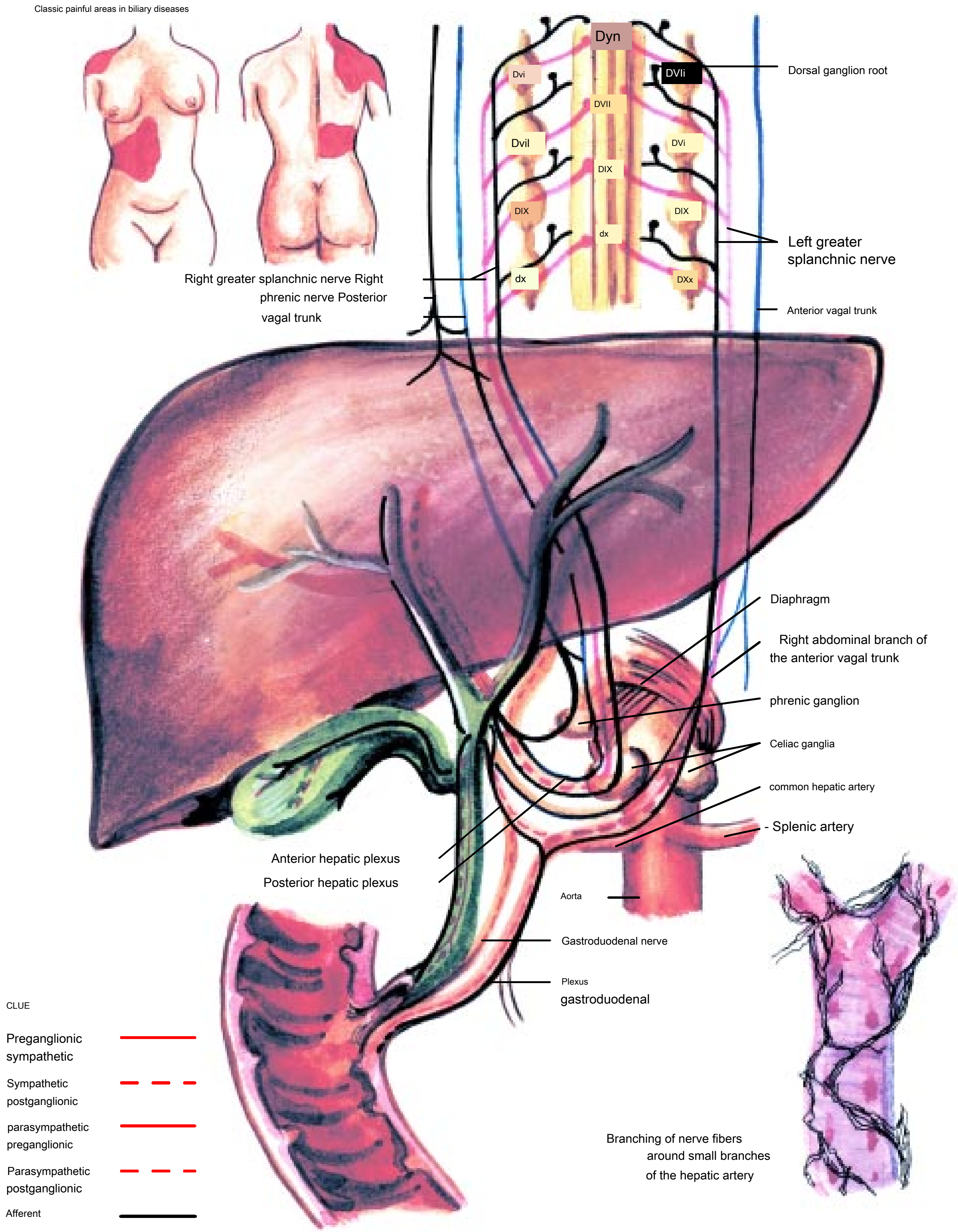

Classic painful areas in biliary diseases
Dyn
Dvi
DVIi
Dorsal ganglion root
DVII
Dvil
DVi
DIX
DIX
DIX
dx
Left greater splanchnic nerve
dx
DXx
Right greater splanchnic nerve Right phrenic nerve Posterior vagal trunk
Anterior vagal trunk
Diaphragm
Right abdominal branch of the anterior vagal trunk
phrenic ganglion
Celiac ganglia
common hepatic artery
- Splenic artery
Anterior hepatic plexus
Posterior hepatic plexus
Aorta
Gastroduodenal nerve
Plexus gastroduodenal
CLUE
Preganglionic sympathetic
Sympathetic postganglionic
parasympathetic preganglionic
Parasympathetic postganglionic
Afferent
Branching of nerve fibers around small branches of the hepatic artery

Fig. 57.9 Pain of biliary origin.

postoperative. We also point out urinary conditions, regional neuralgia, retroperitoneal conditions of the region, psoitis and adnexitis in women. In the particular case of acute appendicitis - a common and essentially surgical condition - the pain generally begins epigastrically, revolves around the navel and settles in the right iliac fossa, with possible radiation to the thigh.

l) Hypogastrium. The pain corresponds to ileitis, sigmoiditis, conditions of the urinary bladder and prostate, and metropathies in women.

m) Left iliac fossa. In that region the pain should correspond to lower left colitis, sigmoiditis, genitourinary conditions on that side, psoas conditions, without discounting regional neuralgia.

n) Location spread throughout the belly. Finally, abdominal pain presents this location, that is, it varies from place to place, with a true tradition along the intestine in the sense of circumference, for the small intestine, or following the colic framework, associated or not with the desire to defecate. This pain, called by the common people " twisting", "squirming" or "colic" itself, corresponds to a violent hyperperistalsis of the small intestine or more frequently the large intestine, which is observed in inflammatory processes of the intestine, particularly in acute colitis and enterocolitis, some tumors, etc.

Although pain in the lower lumbar regions and lumbosacral spine does not generally correspond to a digestive pathology, it is interesting to note that said low posterior pain can also correspond to certain types of enteropathies, retroperitoneal tumors, ileitis and some pancreatopathies. We already mentioned the semiological value of posterior pain of a higher location, when we refer to epigastric pain.

2. Irradiation. As with all the characteristics of abdominal pain, the irradiation and location can be narrated spontaneously in the history of an intelligent and observant patient. The patient gestures and brings his hand and fingers to a precise part of the abdomen, as well as pointing out the direction of irradiation to neighboring regions. On the contrary, in most cases, the doctor is forced to ask the right questions to obtain the most and best information. In this aspect the most useful questions are:

Can you show me with your hand where you feel the pain?

Is your pain fixed in the pit of your stomach, or does it spread?

In this case, where does it run?

Let's analyze the different possibilities of epigastric pain irradiation:

a) Upwards, towards the thorax. Of the anginal type, it corresponds to lesions lower in the esophagus and upper in the stomach, juxtacardial (ulcers), and diaphragm (hernia). Let us also point out the alterations of the coronary artery.

b) Towards the left, to the hypochondrium on that side. Lesions of the pancreas, especially the body and tail, have this radiation.

c) Towards the right. It corresponds, as we have pointed out, when dealing with right-localized pain, hepatovesicular affections, and duodenitis.

d) Downwards, such as towards the navel or rest of the belly. Let us keep in mind the epigastralgia of acute appendicitis and injuries of the ileocecal region, pain that ultimately settles in the right iliac fossa. We will return to this type of pain when studying non-epigastric abdominal pain.
In some cases of perforated ulcer (lesser curvature or anterior face of the duodenal bulb) irradiation can also be to the right iliac fossa.
In general, distal duodenal and proximal jejunal disorders may give this type of irradiation. This irradiation can also be seen in prolapses of the gastric mucosa through the pylorus, especially when there is inflammation (gastritis) of said mucosa.

e) Towards the back, towards the spine and the interscapulovertebral spaces. It corresponds to certain ulcers, small curvature, posterior contact (D5 to D10), duodenal ulcers (D9 to L2), and pancreatic processes; We have already seen during the study of the location that sometimes there is no epigastralgia, but only the posterior pain.

f) Finally, irradiation can be universal, broad. It covers, as the patient points out, different neighboring regions starting from the epigastrium. This occurs in cases of perivisceritis starting from the stomach, duodenum, gallbladder, etc., whether primitive or secondary. In these cases, the pain goes up, to both shoulders, to the rest of the belly, making the patient desperate. In tabes, irradiation in a girdle or belt is indicated, around the belly starting from the epigastrium. Universal irradiation is also indicated in acute peritonitis and pancreatitis. In jejunum processes in general, irradiation part of the mesogastrium and faces upward and to the left. Pain in the ileum tends to radiate downward and to the right or left, depending on the location of the affected loop.

The processes of the terminal ileum, cecum, and appendix, generally located in the right iliac fossa, radiate toward the umbilicus and epigastrium. Radiation to the right thigh or right leg is also observed. Pain in the ascending colon tends to radiate to the left.

Those of the transverse colon, downwards and also to the left. All sigmoid pain radiates downward and to the left, toward the crural region and along the midline. As towards the rectum and perineum, to whose region the pain of rectal origin radiates. Abdominal pain of a neuritic or neuralgic type has the tendency to radiate like intercostal pain, that is, in a metameric sense. The pain is identified at the parietal emergence points.

Due to the extension of the intestine and its multiple curvatures, these indications mean more than a guideline given the multiple variants.

3. Periodicity. Periodicity of abdominal pain is understood as the relationship or evolution of this symptom within a certain time, generally referring to at least one year. Thus we know a type of pain, which within the year occurs to the patient once or twice, during a period of activity, say two or three weeks of pain, more or less daily, alternating with a more or less long period (generally months) of calm or remission.

In turn, periods of activity or painful periods are characterized by being of limited duration and separated by periods of calm, generally of longer duration than those of activity. This type of pain is called periodic pain. On the contrary, there is another type of pain, which behaves throughout the year in a totally different way. Patients present their pain in a more or less constant manner, without any characteristic of alternation, nor more or less interspersed phases of calm. This second modality is called constant onoperiodic pain.

Of course, there may be examples of pain that do not behave clearly in one direction or another, identifying them as pain of atypical periodicity or relative constancy.

In summary, we can say that according to their periodicity, the pains can be:

a) Typical periodic pains. Of more or less long periods, of weeks. The most typical periodic abdominal pain corresponds to pain epigastric ulcer produced by gastric and duodenal ulcers. The painful periods in these cases last two or three weeks, or less frequently four, and up to two months in exceptional cases. Interspersed periods of calm or remission are observed, so clear that the patient believes himself to be completely cured (fig. 57. 10).

It is also interesting to note that the periods of painful activity in ulcers occur particularly during the spring and autumn months. These seasonal relapses are seen more in duodenal ulcer.

The absence of periodicity of epigastric pain or the marked prolongation of a painful period does not deny the possible existence of an ulcer, either due to atypicality or because there is some complication: pyloric stenosis or deepening of an ulcer that reaches the peritoneum; These organic alterations annul the character of periodicity.

In turn, the periodicity of epigastric pain, no matter how typical, should not be considered exclusive to ulcers of the stomach and duodenum. Very frequently, the periodic pains typical in some gastritis, in certain non-ulcer pyloroduodenal syndromes, and in duodenitis, which constitute the pseudoulcerative syndrome, should be noted.

b) Atypical periodic pains of more or less short periods, days (fig. 57.11). Atypical periodic pains are characterized by being the briefest, lasting only two or three days and being separated by intervals of several weeks or months. The observation during the interrogation of this type of periodic pain suggests hepatobiliary processes. These pains also occur suddenly, especially in gallstones, causing the so-called biliary colic; They are triggered by a change in diet (diet overloaded with fats), an uneven trip or an emotion due to disgust. They are more frequently observed in women and in relation to their periods, lactation, pregnancy or other genital crisis, which demonstrates the hormonal-metabolic influence on these processes.

c) Non-periodic pain:

- Pains with regular duration throughout the year. These pains, which are reported by patients as constant, which they suffer for years from a moment

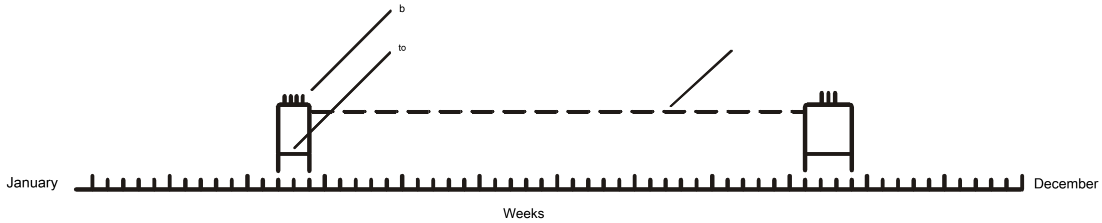


Fig. 57.10 Schematic conception ("gastric year") of the periodic pains typical of gastric ulcers and especially duodenal ulcers:

a, period of painful activity; b, painful crisis during period;

c, calm phase or absence of pain.

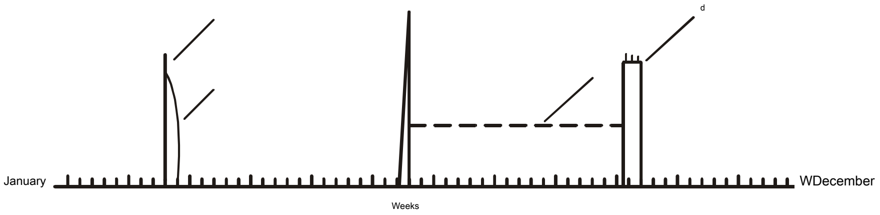


Fig. 57.11 Schematic conception ("biliary year") of the atypical periodic pains of cholecystopathies, especially due to lithiasis (biliary colic):

a, painful period; b, painful crisis; c, calm phase; d, painful crisis.

determined, generally correspond to pathological processes of the stomach, duodenum and other viscera of the upper abdomen, to which extravisceral factors are added. Here, the Morley reflex or peritoneosensitivVo is the rule. We point out as typical examples:

Secondary perivisceritis, either due to old inflammatory processes or due to surgical adhesions. Let's mention periappendicitis, perityphlitis, periduodenitis, among others. The conjunctive reaction, violent in these cases, irritates the nervous system, determining the sympathetic and somatic reflex at the same time. It is clear that since the cause of the reflex is a permanent irritating thorn and peritoneal contact, the pain will be constant and there will be no periods of calm or remission.

Chronic ulcers and tumors, especially cancer in the externalizing phase.

In cases of chronic ulcers with a covered perforation, the characteristics of the patient' s pain will change and they will have more or less constant pain throughout their gastric year. Primitive perivisceritis, which is rarer, but may be determined by the

tuberculosis.

Primitively extradigestive organic processes, dependent on the central nervous system (medullary, radicular, vertebral, etc., where epigastric somatic pain also has characteristics of permanence without any periodicity.

Pains with irregular permanence throughout the year. Let us note, finally, among the non-periodic epigastric pains, the irregular pains of psychoneurotic patients.

In non-epigastric abdominal pain, the phenomenon of periodicity is far from being as clear as in epigastric pain, but it can be used to assess those pathogenic factors related to climatological, epidemic, allergic, etc. factors, which necessarily explain the alternating nature of many abdominal pains. However, sometimes abdominal pain with a more or less typical periodicity is observed in some allergic processes of the intestine, pain that depends on the cyclical nature of this pathology. Periodic pain may occur in so-called abdominal epilepsy and in some forms of tabes. Some types of diverticulosis can also cause periodic pain, due to the addition of inflammatory pressure.

4. Rhythm or schedule. The rhythm or schedule of abdominal pain is understood to be the relationship or evolution of this symptom within a period of 24 hours. In general, epigastric pain can have a defined relationship within the day with meals or prandial phases (what is known as "gastric day", "dyspeptic day" by Soupault and Gutmann), or, on the contrary, it can occur during the day without the slightest prandial or time relationship. We can easily deduce two fundamental types of epigastric pain:

a) Epigastric pain with rhythm, schedule and meal relationship:

- Early or early postprandial pain. It is one that occurs almost immediately after the patient has eaten. He eats and the pain appears for a certain time, the pain can disappear and reappear in similar circumstances after another meal. Early static pain (almost always occurs due to organic lesions), can correspond to gastritis and occurs when food comes into contact with an inflamed mucosa, in cases of prolapse of the gastric mucosa through the pylorus, in tumors or other organic processes that reduce gastric capacity and can also be caused by stretching of the solar plexuses in cases of ptosis or other anomalies of shape and position. (dolichogastria).

Late or late postprandial pain. It is one that occurs 1-5 hours or more after the end of the meal. The patient eats, feels very well, and within the digestive period, more or less late, begins to feel pain, which lasts longer than the early type of pain. The mechanism of late pain production is essentially pathophysiological or dynamic and is closely related to peristalsis, motility and stomach emptying. In general, late pain is relieved by emptying the stomach or eating again, as opposed to early pain that is exacerbated by eating more food. Late pain is also the expression of an altered motor function, of the stomach's fight against a more or less permanent obstacle that prevents its normal evacuation. It is a pain, in short, essentially gastric, digestive.

These pathophysiological considerations regarding delayed pain facilitate the understanding of its diagnostic value. Late pains correspond to ulcers of the stomach and duodenum (juxtapyloric). Although in ulcers, especially near the pylorus, the late painful rhythm that occurs at the third or fourth hour is very common, we must also think that this painful rhythm can also occur in any other juxtapyloric process and sometimes even in distant processes. In any case, when faced with delayed pain we will preferably think of low-lying ulcers, particularly in the duodenal bulb, in which case the pain occurs 3-5 hours after meals, except in cases of pyloric obstruction, which sometimes produce pain.

ultra-late periods accompanied by evacuation vomiting. In ulcerative disease, the pain may be accompanied by a feeling of hunger (painful hunger or gastralgocenosis) . The fact that it subsides after a new intake clearly separates it from prandial pain, which is triggered when eating food. Afterwards, cholecystopathies should be considered, in which case the pain generally occurs later than in duodenal ulcer.

Hepatic colic, whose expression of location, irradiation and periodicity we already know, has a start time, in the early morning (1:00-2:00 a.m.) for people who eat between 7:30 and 8:00 p.m.; That is, its late appearance is 6-8 hours after the afternoon meal, since it is not frequent in relation to the midday meal, that is, our lunch (12:00 m.-1:00 p.m.).

Duodenal ulcer patients also present with nocturnal pain that is relieved by eating. We will conclude the study of the type of epigastric pain with a more or less defined prandial rhythm or schedule, with the presentation of the special ulcerative rhythms (Table 57.2), described by Moyniham, an English surgeon, pioneer of these studies, although they are currently accepted only as a guide.

Table 57.2

<table>
<tr><th>Four-beat rhythm of | gastric wax aul</th></tr>
<tr><td>1. Food
2. Pain relief
3. Early recurrence of pain
4. New (spontaneous) calm of pain</td></tr>
<tr><th>Three-beat rhythm of I aúl duodenal wax</th></tr>
<tr><td>1. Food
2. Pain relief
3. Late recurrence of pain</td></tr>
</table>

b) Epigastric pain without rhythm and without defined prandial relationship. It constitutes continuous pain and irregular pain, within the 24-hour gastric day cycle:

Continuous epigastric pain. In reality, it is not gastric pain, or even digestive pain, but rather extradigestive. It is a somatic pain, due to Morley's peritoneal-sensory type reflex with direct participation of the cerebrospinal system. Patients report that they suffer pain hours after hours, throughout the day and night, with a permanence that sometimes makes them desperate; This pain is sometimes exacerbated. The most typical example of continuous pain is that produced by perivisceritis corresponding to ulcers or tumors that infiltrate the peritone0, as well as any process located in the metameres of the epigastrium that affects the somatic nervous structures of the region; root, spinal, vertebral, anterior wall injuries, etc.

tera.

Irregular pain. It occurs within 24 hours, during which the patient may report it in the morning, afternoon or evening;

lasts minutes or hours; It may or may not be related to food; In short, it is a pain with a capricious course on the gastric day."

This type of pain corresponds fundamentally to painful dyskinesias of psychovegetative cause and is observed in psychoneuroses and neurovegetative dystonias.

When referring to the rhythm or schedule of pain in the other regions of the abdomen that is not the epigastrium, it does not follow such rich guidelines, but nevertheless requires a good analysis, which results in its diagnostic value.

We must make the effort to reconstruct the "patient's intestinal day":

a) Abdominal pain with rhythm or schedule and meal relationship:

Early or early postprandial pain. It is produced by the pathological exaggeration of the gastrocolic reflex, it appears as soon as the patient eats food, as occurs in most colopathies, particularly in colitis of various origins, and sometimes in colic dyskinesia, it can be accompanied by diarrhea. It is curious to also note the early abdominal pain in the aerocolia of the left angle, in which case the distention of the stomach determines the abdominal pain located in the left hypochondrium and the expulsion of gas through the anus.

- Late or late postprandial pain. This pain appears 2 to 6 or 8 hours after meals, of course, the time being related to gastrointestinal transit that is relatively less late for the processes of the jejunum and ileum, and later for the processes of the colon. Let us point out the late abdominal pain of ileitis; and also the pain of the fourth to fifth hour of typhlitis or typhlitis, which corresponds to a process of irritability of the cecum, when the intestinal contents are established at that level. Even later are some pains produced in a limited area of the colon (angle splenic or descending), sometimes nocturnal. In general, these abdominal pains are sometimes intermittent in nature.

b) Pain without rhythm or schedule, or prandial relationship.

They are the most frequent, since the determining causes have nothing to do with the food intake cycle. Most abdominal pain falls into this group. All inflammatory, neoplastic, reflexogenic factors, etc., are generally permanent throughout the day, occurring at any time and without a necessary mealtime relationship, although they may be a cause of exacerbation of gastric filling.

In particular, we can mention as permanent pains within the day, those produced by malignant tumors, perivisceritis, radiculitis.

The abdominal pains of dyskinesias of psychoneurovegetative origin are also constant throughout the day, although capriciously.

To provide the best information regarding the periodicity and rhythm of epigastric pain, especially if the spontaneous history is not sufficient, let us look at the usual form of our questions:

How long have you been suffering from your pain?

Throughout the years of your illness, has your pain occurred continuously, or, on the contrary, has it occurred seasonally, alternating with periods of complete calm?

When do you have your pain crisis? During the day or at night?

Is your pain related to eating?

How does the pain occur throughout the day?

5. Intensity. The intensity of the pain is another highly semiographically oriented character. This factor is conditioned by the psychosomatic background of each patient, and the patient may complain of pain of varying intensity: large, medium and low. It is generally noted that organic processes tend to produce more intense reflections than those of a functional nature. A parallel should not be established between the prognosis and the intensity of the pain, since sometimes a spastic and transient alteration causes more intense pain than a malignant tumor. Let us also remember that there are so-called silent areas, for example the body of the stomach, that are poorly reflexogenic and on the contrary, whenever the painful reflex is due to arterial spasm, more intense pain will occur.

Let's analyze the different types of pain by their intensity:

a) Pains of great intensity. They are the ones who require the use of painkillers to calm down. Let us cite, as examples of highly intense epigastric pain, hepatic colic from gallbladder lithiasis, pain from acute abdominal processes such as perforated ulcer, and acute pancreatitis. Perivisceritis, syphilitic vertebral processes and spinal processes, intestinal obstruction, torsion of a pedicle, strangulation, volvulus, perforations and peritonitis are examples of high intensity non-epigastric abdominal pain. Also particularly intense are the abdominal pains of tabes, lead poisoning (saturnine colic) and mesenteric thrombosis.

b) Medium intensity pain. Gastric and duodenal ulcers, as well as non-calculous cholecystopathies, are typical examples of medium intensity pain located in the epigastrium. In the rest of the abdomen, the pains of motor dyskinesia of the colon, some enteritis (idiopathic ulcerative colitis, amoebic, etc.), and tumors are located.

c) Low intensity pain. They are sometimes cited by patients as epigastric discomfort, as a feeling of sorrow in said region; They correspond to gastric dyskinesias of different kinds, some types of gastritis and duodenitis, etc.

6. Quality or character Many times spontaneously or through questioning, patients detail not only the intensity of their pain, but how they feel it;

that is, its specific quality. Many qualities of pain are distinguished, some difficult to identify and interpret because pain is a subjective quality, felt and expressed differently according to each patient. Below are the main types:

a) Burning or burning pain. It is identified by the patient as burning, burning pain.

Among the diseases that can cause this type of pain in the epigastrium we must mention gastritis, in those processes that are accompanied by bile reflux into the stomach. This burning sensation is also produced by the contact of the irritating intestinal contents with the altered colic mucosa; in typhlitis and typflectasis with fermentative syndromes.

b) Cramp or twisting type pain. It is identified by the sensation of painful contracture experienced by the patient, which is expressed as coic or cramping pain. Pathophysiologically, this modality implies a dynamic phenomenon with the presence of parcel spasms or areas of achalasia. Thus a factor of vigorous peristalsis, of struggle, is established; The more or less abrupt stoppage of a peristaltic wave is observed (fluoroscopic verification) which coincides in most cases with an alteration of gastric evacuation. It is observed in gastric and duodenal ulcers and cholecystopathies.

In enterocolic inflammatory processes of different origin, in tumors with an obstructive tendency, as well as in many dyskinesias; It is very evident in colopathies.

c) Grave pain. It is identified as pain with a sensation of heaviness, of a certain mechanical distension of the gastric organ. Its production mechanism responds in many cases to a hypotonic or atonic alteration of the stomach with poor evacuation. The possible intervention of the abdominal wall due to relaxation, causing poor support, as well as stretching of the nervous plexuses, should also be noted. In these cases, gastric ptosis is considered, generally associated with neurovegetative vasomotor alterations, antral gastritis and duodenitis, and hypotonic or atonic dilation of the intestine or colon.

d) Terrible pain. Expressed by the patient as something not only intense, but also with a sensation of depth, of penetration, as if produced by a drill. It corresponds to processes with marked peritoneal participation (Morley's reflex), which in their production mechanism leave the neurovegetative system to fall into the cerebrospinal sphere, the use of strong analgesics being almost always necessary to calm them. Its location in the epigastrium is observed in perforating ulcers in the pancreas and in malignant tumor infiltrations, among other diseases.

e) Agonizing or excruciating pain. The sensation is something greater than the psychic resistance of the patient. It is produced by the intervention of an arterial spasm, recalling the mechanism of the pain of coronary or anginal processes.

It is observed in some cases of hepatic colic due to gallstones with common bile duct intervention, in gangrene of the gallbladder and in abdominal angina.

f) Lancinating pain. As its name expresses, it is a painful sensation of lancet shots, sudden and sharp pains, fortunately of short duration with a certain latency phase. It is observed in pain due to organic nervous alteration, as in tabes, and in lead colic, among the most typical examples.

g) Flashing pain. It is very fast; corresponds to sudden spastic phenomena.

h) Constrictive pain. Sensation of retraction, of pressure, as occurs in cases of parieto-abdominal neuralgia and herpes zoster.

i) Dull pain. It is of low intensity, but more or less constant, imprecise, in which cases the inciting reflex, although permanent, is of poor performance. This is observed in many cases of digestive ptosis, and in some cases of gastritis.

) Throbbing pain. With throbbing sensation; It corresponds to the intervention of large caliber vessels, as observed in the aneurysms and in the aortism (padrejón) of many neurovegetative dystonic patients.

k) Pain with tenesmus and pushing. Tenesmus consists of a burning sensation and peritoneal weight, with a constant desire to defecate without being able to do so. It is a continuous and sterile desire. The patient first defecates one or several times and then the pain remains in the form of tenesmus. Or on the contrary (the association between both symptoms is common), the patient perceives the pain with straining, a sensation of involuntary contraction of the rectum without excreting nothing. Pushing constitutes an unsuccessful spasmodic phenomenon, with a foreign body sensation. Both symptoms, straining and tenesmus, are present in rectosigmoid or dysenteric processes of any etiology.

7. Start mode:

a) Sudden onset pain. It occurs in most acute abdominal pain, for example, in perforated ulcer, in acute pancreatitis, in appendicitis and in hepatic colic. Severe dysenteric infections, acute poisoning, surgical injuries (intestinal perforation, occlusion due to strangulation, among others) (fig. 57.12).

b) Slow onset pain. In most cases of uncomplicated gastric and duodenal ulcers and especially in dyskinesias, the onset of pain is slower. Such is the case of irritable bowel syndrome (Fig. 57.13).

8. Way to calm down. There are pains that are relieved by the ingestion of food or antacids, as occurs with the so-called epigastric hunger pain, corresponding to most typical duodenal ulcers. The mechanism of production of this type of pain is almost always explained by the

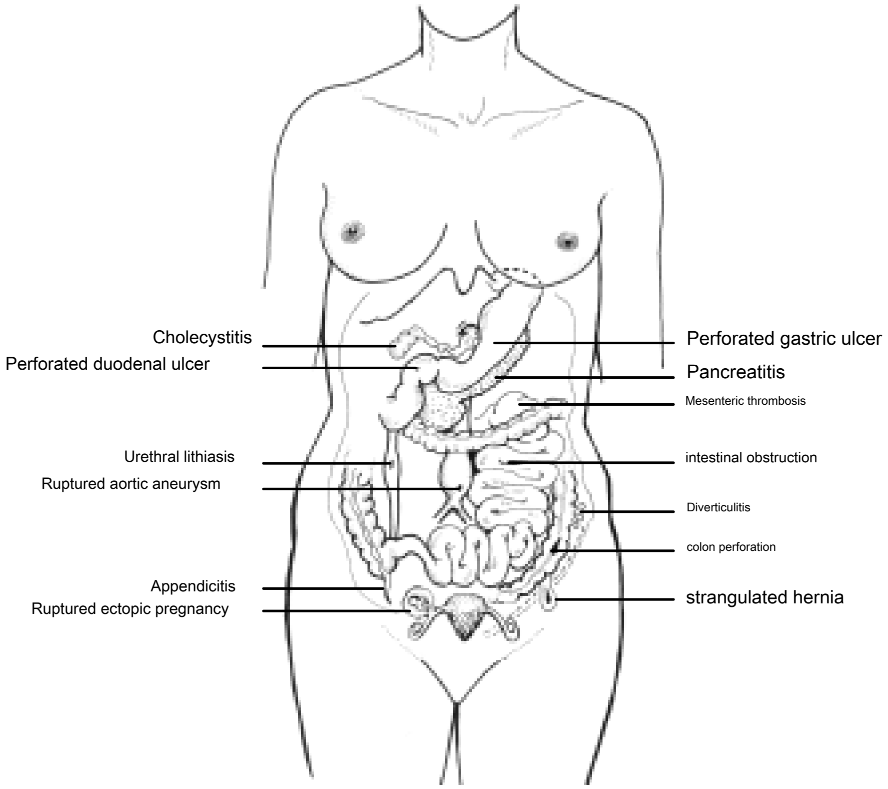
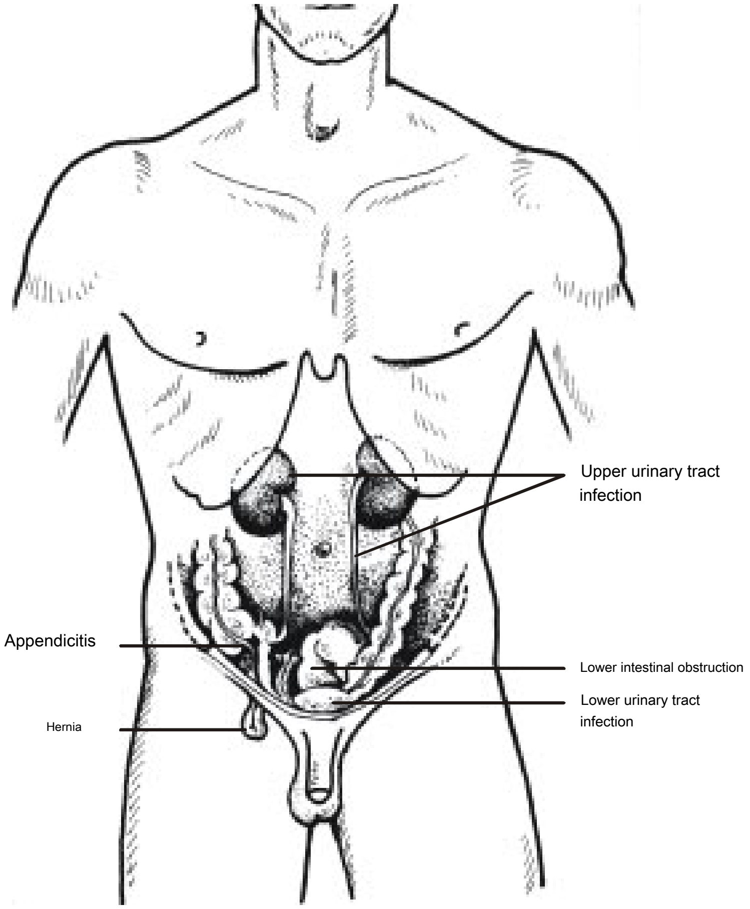

Cholecystitis
Perforated duodenal ulcer
Urethral lithiasis
Ruptured aortic aneurysm
Appendicitis
Ruptured ectopic pregnancy
Perforated gastric ulcer
Pancreatitis
Mesenteric thrombosis
intestinal obstruction
Diverticulitis
colon perforation
strangulated hernia

Fig. 57.12 Common causes of abrupt onset abdominal pain.

Fig. 57.13 Common causes of slow-onset abdominal pain.


Upper urinary tract infection
Appendicitis
Lower intestinal obstruction
Lower urinary tract infection
Hernia

existence of hypersecretion with hyperchlorhydria, and relief is achieved by neutralizing excess acidity with milk or an alkaline substance. There are pains that require the administration of an antispasmodic medication, which shows that they are caused by spasm and poor evacuation; In these cases, vomiting calms the pain as the spasm disappears. Let us cite as examples the pain of juxtapyloric ulcers and cholecystopathies, among others. Finally, there are pains that require calming, analgesic medication, as occurs in biliary colic, perivisceritis, spinal cord involvement, etc. In general, when the cerebrospinal system intervenes, analgesic medication is almost always indispensable, which can go as far as nerve blockade.

Recumbent relief relieves pain from dolichogastria, solar plexus injuries, and dumping syndrome (jejunal hyperosmotic syndrome sometimes seen after gastrectomy); On the other hand, it aggravates or initiates the pain of the hiatal hernia, while standing and walking relieve it.

9. Variation according to position changes. It is of great semiological value to verify in the patient's narration how they observe the variation of pain (increase or decrease) according to the position adopted. have. For example, in perivisceritis it is found that the patient knows the appropriate position to relieve his pain, which is explained by the decrease in tension or stretching of the nervous plexuses due to the ties or adhesions. In uncomplicated ulcers and cholecystopathies, this relationship is not appreciated. In gastric ptosis we also see how the patient improves his painful discomfort by lying on a bed with the skin raised or by suspending the belly with his hands or with a suitable belt. We will insist on the position, when we deal, in the physical examination, with the study of the maneuvers and the signs related to these events.

10. Associated symptoms. It is essential to collect, in parallel with the pain, the symptoms of intimate or simultaneous presence, since they will guide the diagnosis of abdominal pain.

If epigastric pain is associated with vomiting and hematemesis, gastric ulcer, duodenal ulcer and cancer may very well be considered. The presence of vomiting forces us to take into account processes with poor gastric or juxtapyloric evacuation, and the association with hematemesis suggests processes of vascular alteration or a blood dyscrasia.

The association of pain with neurovegetative symptoms, such as palpitations, anxiety, nervousness and vasomotor alterations, indicates the possible presence of psychovegetative or hormonal dyskinesias as a cause.

The association of jaundice with pain leads to the development of hepatobiliary processes. If jaundice has immediately followed hepatic colicky pain, there is a strong possibility that a gallstone involving the common bile duct may be present.

Constipation indicates the existence of an arrest in fecal transit with fighting hyperperistalsis; Intestinal obstruction will be considered, due to volvulus, invagination, strangulated hernia, tumors, as the main examples. There may also be a transient spastic occlusion.

If there is associated diarrhea, it indicates the existence of an inflammatory, toxic or infectious process. Examples are amoebic and bacillary dysentery, ulcerative colitis and colonic dyskinesia. There are also tumors that cause pain with associated diarrhea, as occurs in many tumors of the right colon.

Melena and enterorrhagia indicate that the pain is produced by a hemorrhagic process, possibly ulcerative or tumorous, although without excluding other conditions that cause hemorrhage; For example, mesenteric thrombosis and intestinal intussusception in children are reported.

Other associated symptoms may be abdominal distention, borborygmi and colic heartburn, which have a very broad significance when associated with pain and raise multiple possibilities.

11. Course. This factor summarizes the set of the ten factors previously studied. Overall, we can classify epigastric pain according to its semiographic characteristics, into two main types:

a) Regular pain. They have more or less defined characters and a certain semiographic precision, which facilitates their identification. This type corresponds, in general, to organic processes. The characteristics of each case will guide the differential diagnosis: ulcer, cholecystopathy, perivisceritis, tumors, etc.

b) Pains of irregular course. Their characteristics cannot be specified in them and the identification of the ten factors previously indicated is almost impossible. These pains, with a capricious course and evolution in every sense, should always make one think of psychovegetative, hormonal, even essential or idiopathic processes, since their production mechanism is far from being defined and constant.

To provide the best information regarding the periodicity and rhythm of epigastric pain, especially if the spontaneous history is not sufficient, let's look at the usual form of our questions:

How long have you been suffering from your pain?

Throughout the years of your illness, has your pain occurred continuously or, on the contrary, has it occurred seasonally, alternating with periods of complete calm?

When do you have your pain crisis? During the day or at night?

Is your pain related to eating?

How does the pain occur throughout the day?

VOMIT

This symptom is the second most important in the triad of major symptoms or capital symptoms of the gastroduodenohepatobiliopancreatic segment.

Concept

Vomiting or emesis should be defined as the rapid and strong expulsion of gastric contents to the outside, accompanied or not by nausea and other satellite symptoms. This definition is intended to include the two fundamental types of vomiting: peripheral and central; the first of a reflexogenic nature, and the second produced by the direct stimulation of the vomiting centers.

Peripheral vomiting is identified by the presence of the symptom nausea as a phenomenon prior to vomiting. In central vomiting, nausea and other satellite symptoms are not observed, the symptom being limited to the expulsion of gastric contents. Vomiting should not be confused with gastric regurgitation, a dyskinetic type symptom.

The terms "throwing" and "provoking" are indicated by the common people as equivalents of vomiting, and the term " arqueadas" as equivalent of nausea.

Vomiting has great semiological importance. Its frequency is enormous and its semiographic valorization is very difficult due to the innumerable causes that can produce it.

Semiogenesis or pathophysiology (fig. 57.14)

Peripheral vomiting

It is produced by the stimulation of the so-called vomiting reflex. As in any reflex arc, the afferent or centripetal pathways must be distinguished, fundamentally represented by the vagus and the sympathetic and, in an accessory order, by the vestibular, glossopharyngeal, optic and nerve nerves.

olfactory. The efferent or centrifugal pathways are represented by the vagus and sympathetic nerves, as well as by the phrenic nerve, the spinal nerves and the cranial nerves that innervate the pharynx, soft palate and epiglottis.

The vonnitive centers are located in the medulla and are neighbors of the dorsal nucleus of the vagus. They are also described in other points on the floor of the fourth ventricle: cruciform sulcus, restiform bodies, quadrigeminal tubercles and striate bodies.

The destruction of these centers results in the loss of this reflex, and its slightest excitation with apomorphine causes vomiting; These experimental facts prove the validity of this location. Any stimulation at different levels of the digestive system or outside it, conducted by the vagosympathetic endings, may trigger the reflex and cause peripheral vomiting. According to the law of the distance of irritating spines, mentioned so many times, vomiting will occur more easily in cases of irritating spines close to the stomach.

central vomiting

In this type of vomiting, the production mechanism is limited to the direct stimulation of the emetic centers described, as occurs in organic lesions of the central nervous system that increase intracranial pressure (tumors, abscesses, meningitis, meningeal hemorrhage, etc.) or its stimulation through the blood caused by various substances with emetic action, whether exogenous (ipecac, apomorphine) or endogenous (uremia, acetonemia).

Sometimes vomiting is caused by psychic influences (nauseous smell, disgusting taste, etc.).

Intimate mechanism of gastric emptying in vomiting

It has been well studied by Cannon, who reproduced it experimentally in the cat, causing central vomiting by injecting apomorphine. This author observed a complex series of movements, which he described neatly as follows:

At the beginning, relaxation of the upper portions of the stomach is observed, as well as the cardia up to its esophageal side. This is followed by one or more deep peristaltic waves passing from the middle portion or body of the stomach towards the incisura angularis, at which level they apparently terminate or continue very weakly towards the pylorus. A vigorous contraction is then formed at the level of the incisura angularis that divides the stomach into two cavities, preventing the gastric contents from passing downwards, to the pyloric antrum and the duodenum.

This is followed by a strong contraction of the diaphragm and abdominal muscles with elevation of pressure. intra-abdominal sion. This pressure, acting on the upper cavity of the stomach (in perfect relaxation) determines the expulsion of its contents upward, which comes out through the mouth to the outside. As can be seen, the stomach plays a passive role in this last process by fundamentally acting on the factors of the extragastric muscles. In fluoroscopic observation, antiperistalsis of the pylorus toward the antrum was rarely detected.

During the expulsion of gastric contents, the esophagus remains relaxed, the glottis closes, respirations are inhibited, the laryngeal cartilages project forward and become rigid. The pharynx expands, the soft palate folds upward, thus preventing vomited material from entering the nasopharynx (fig. 57.15).

In man, these phenomena occur in a similar way, antiperistalsis is rarely observed, which is established only in cases of infragastric intestinal obstacle, in which the intestinal contents (bile or jejunoileal contents) previously reflux to the stomach and vomiting subsequently occurs. It is also interesting to note, according to Hatcher and Weiss, the important role that relaxation of the cardia plays in the greater or lesser ease of vomiting in different subjects.

All these phenomena are coordinated by the vomiting centers, markedly influencing the neurovegetative condition of the subject. The threshold of the vomiting center is specific to each individual, so there is a different reaction to the same stimulus, and some people vomit more easily than others.

Magendie verified the passive role of the stomach in vomiting by causing this symptom experimentally in the animal, after resecting the stomach and replacing it with a water bladder. Gianuzzi verifies the fundamental role of the abdominal muscles in preventing vomiting in a cured animal (muscular paralysis due to curare).

Semiography and semidiagnosis

Clinical characteristics of peripheral and central vomiting

Peripheral vomiting is characterized by the expulsion of gastric contents accompanied by nausea and marked satellite symptoms. In the act of vomiting, the patient experiences an unpleasant sensation of great discomfort, dizziness, anxiety, epigastric emptiness with cold sweats, tachycardia, respiratory arrhythmia, carrying out abdominal contractions that determine the violent exit, through the mouth, of the gastric contents. Vomiting may occur one or several times until the stomach is emptied, or the phenomenon may even persist after emptying the stomach. Nausea, which precedes vomiting, can persist and last for a certain time with algidness and sometimes with lipothymia.

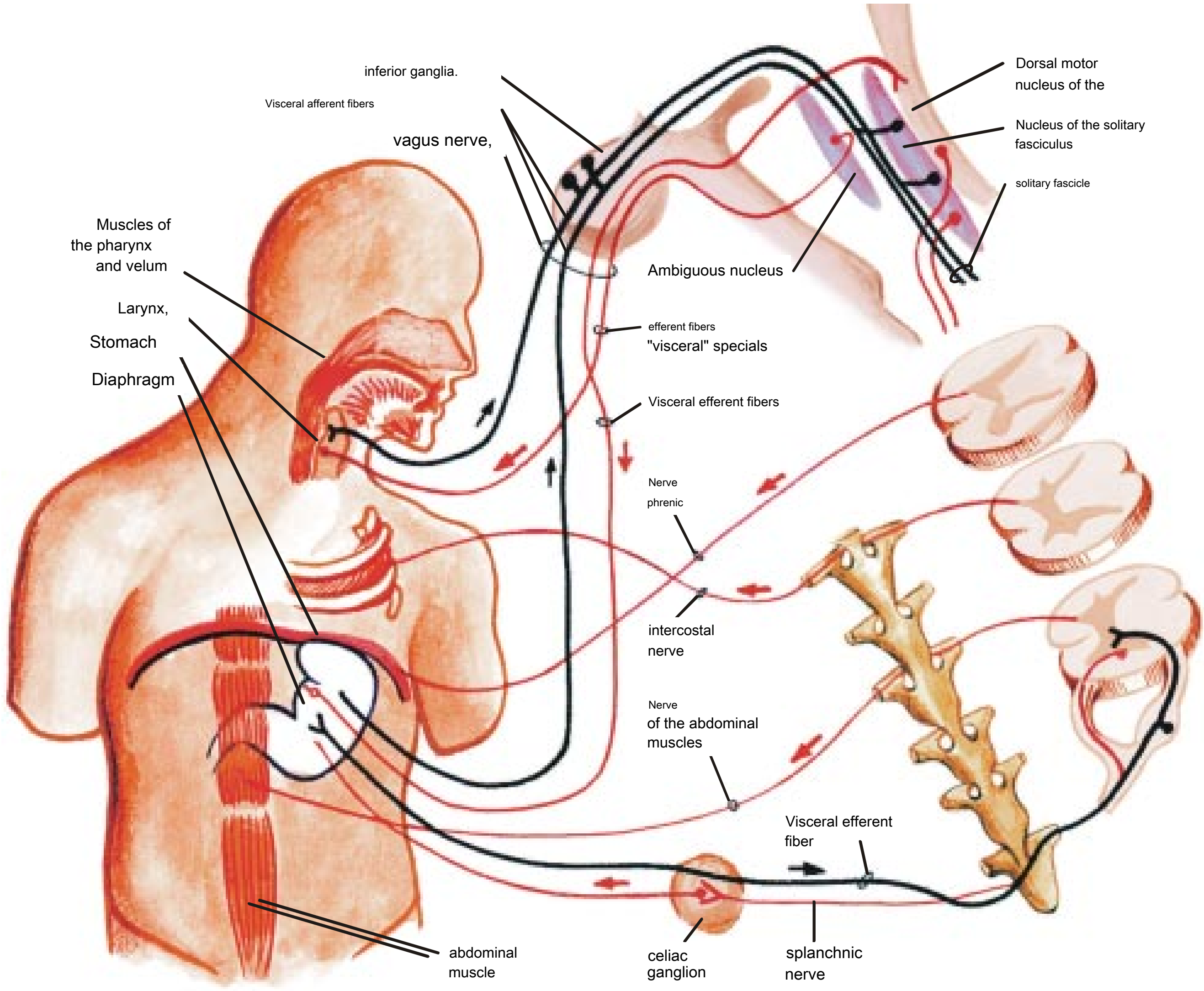

inferior ganglia.
Visceral afferent fibers
vagus nerve,
Dorsal motor
nucleus of the
Nucleus of the solitary
fasciculus
solitary fascicle
Muscles of
the pharynx
and velum
Ambiguous nucleus
Larynx,
Stomach
Diaphragm
efferent fibers
"visceral" specials
Visceral efferent fibers
Nerve
phrenic
intercostal
nerve
Nerve
of the abdominal
muscles
Visceral efferent
fiber
abdominal
muscle
celiac
ganglion
splanchnic
nerve

Fig. 57.14 Semiogenesis of vomiting.

Central vomiting is characterized by the expulsion of gastric contents without nausea or other relevant satellite symptoms. The patient expels the gastric contents in the form of a "spindle" or "projectile", sometimes more or less insensitively.

Let us make a general list that serves as a guideline, even if it is incomplete, of the main diseases capable of producing vomiting, whether peripheral or central.

Peripheral vomiting can be observed mainly in various digestive diseases: gastric and duodenal ulcers with pyloric alteration, cholecystopathies, liver disease, pancreatopathies, gastroduodenitis, appendicitis, intestinal obstruction, digestive dyskinesias. neurovegetative and psychoendocrine, etc. It is also observed in conditions of other systems and in motion sickness (motion sickness): this relationship becomes endless, since any irritating thorn, at a greater or lesser distance, can produce reflex or peripheral vomiting, always subject to the law of distance.

Central vomiting can be observed by a mechanism of exogenous central excitation: poisoning by emetine, apomorphine, emetic tartar, lobeline, etc. Of endogenous cause, let us point out the central vomiting of cranial hypertension with its multiple causes.

As with all symptoms, careful notation of its particular characteristics greatly guides the causal diagnosis. The most important factors

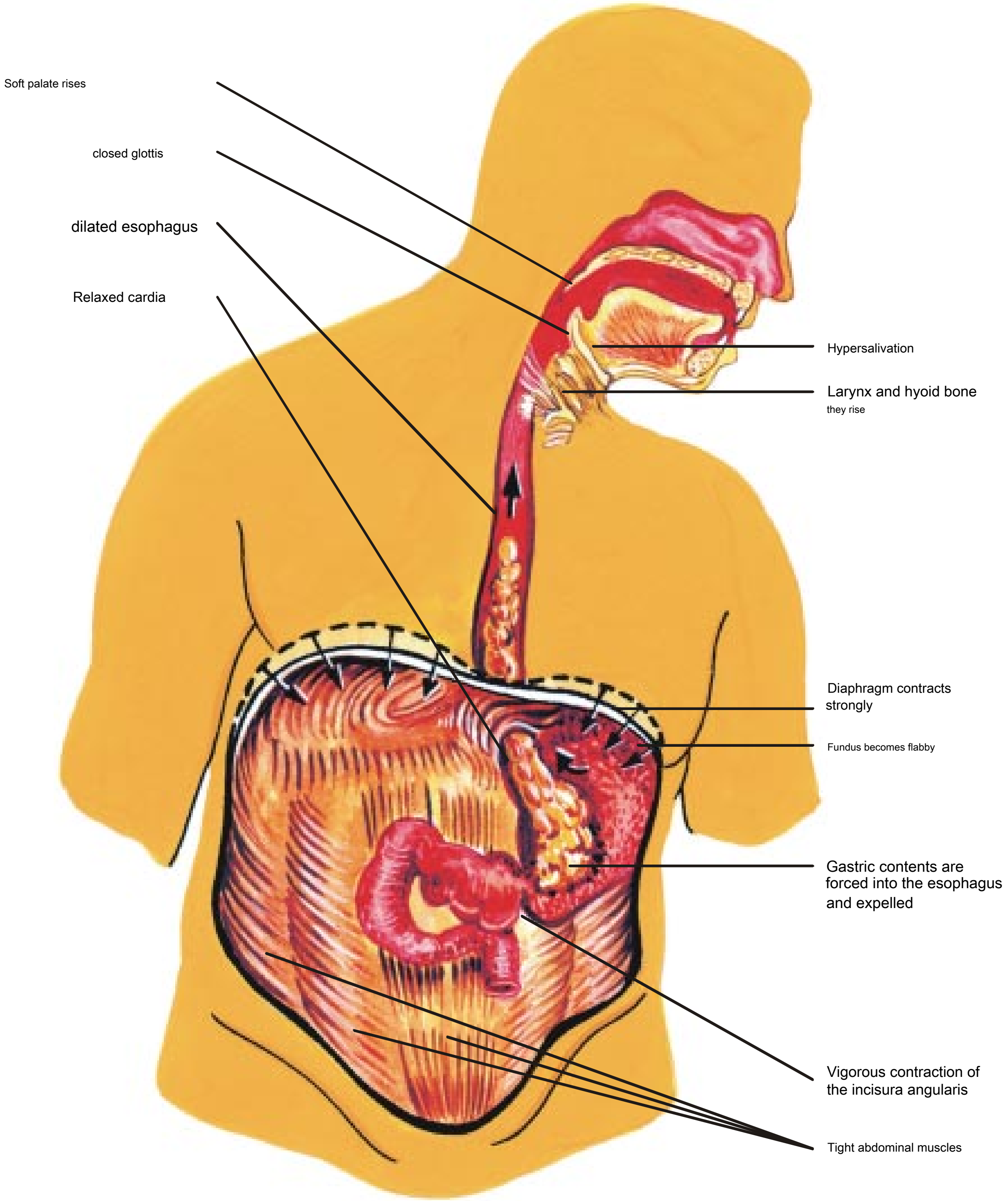

Soft palate rises
closed glottis
dilated esophagus
Relaxed cardia
Hypersalivation
Larynx and hyoid bone
they rise
Diaphragm contracts
strongly
Fundus becomes flabby
Gastric contents are
forced into the esophagus
and expelled
Vigorous contraction of
the incisura angularis
Tight abdominal muscles

Fig. 57.15 Intimate mechanism of gastric emptying in vomiting.

Table 57.3

Semiographic characteristics of vomiting
1. Periodicity 2. Rhythm or schedule 3. Intensity 4. Quality 5. Start mode 6. Way to calm down 7. Associated symptoms 8. Course

Important to analyze in the characterological study of vomiting (listed in Table 57.3) are:

1. Periodicity. In most cases, vomiting does not have any periodicity.

2. Rhythm or schedule:

a) Vomiting with rhythm or schedule and prandial relationship:

Prandial or early postprandial vomiting. It can be presented at the table itself, while the patient is eating or shortly after finishing. The majority of early vomiting is functional, of a reflex nature (cortico-vegetative), and is observed in young subjects, preferably in young girls with ovarian disorders with psychoneurosis of anxiety or anguish, as well as in pregnant women in whom they can be intolerable, being called uncontrollable vomiting.

It is also observed less frequently in older subjects. In general, this early postprandial vomiting is benign, since it is not vomited at all meals, nor all food; On occasions it becomes serious due to its intensity.

Let us also point out the early vomiting of some types of gastritis, in which the initial contact of food with the altered mucosa produces burning, burning pain, and sometimes even reflex vomiting, which is also early.

Let us mention, although they have less diagnostic value because the primary disease is generally known, early prandial vomiting due to vagal irritability, in active pulmonary tuberculosis, vomiting that is manifested by the well-known French cycle: food-cough-vomiting-cough (Piery).

-Late postprandial vomiting. Late postprandial vomiting occurs at variable periods of 1-4 hours after meals. This vomiting depends closely on alterations in the digestion and gastric evacuation cycle. This vomiting of food or digestive type itself, is generally associated with pain, which subsides when vomit; It is established as a consequence of the arrest of peristalsis at low levels of the stomach. For this reason, it is assumed that in most cases there is an irritating lesion or spine, either intrinsic or extrinsic, located in the vicinity of the pylorus.

It is easy to deduce that diseases that cause delayed vomiting will preferably be organic. Let us mention among them low-lying gastric ulcers and duodenal ulcers, especially when these processes are in relatively advanced phases or pylorization phase, as we say in daily clinical language. It is also caused by stomach cancer or other tumors near the pylorus and other less important conditions.

Ultralate postprandial vomiting. It sometimes occurs 24, 48 and 72 hours after ingestion of food. The patient identifies in the vomit, foods that he had eaten two and three days before. In these cases, the obstruction of the pylorus is generally organic and very advanced and sometimes a phenomenon of true gastric asystole is seen, with great dilation, which sometimes makes gastric intubation necessary to obtain complete emptying of the stomach. It is seen in very old juxtapyloric and duodenal ulcers complicated with pyloric obstruction. It can also be observed in cancer or other rarer lesions, of an obstructive, organic, prolonged nature.

b) Vomiting without rhythm or prandial relationship:

Vomiting on an empty stomach. Of great semiological interest due to its relative frequency. Patients report that upon waking up or rinsing their mouth, moments after getting up, they vomit, generally mucous, watery or, more frequently, bilious.

The vomiting of pregnant women is designated as vomiting on an empty stomach, as it tends to occur more frequently at that time. Also, those corresponding to hepatobiliary conditions due to bile reflux to the stomach, as well as gastritis, especially due to alcohol or tobacco, where vomiting is associated with the pituitary. It is also observed in toxaemia, for example, in uremia.

Nonprandial vomiting (at any time). In this group we could include central vomiting (tumors, cranial hypertension, etc.), without any relationship with the digestive cycle. Let us also mention vomiting due to alterations in other systems, which are reflex in nature from irritating spines located at a greater or lesser distance:

circulatory, respiratory, urinary and especially genital. Finally, the functional vomiting of psychoneurosis with gastric dyskinesias appears irregularly and capriciously at any time of the day or night.

3. Intensity. There are types of vomiting that are sometimes so frequent that they seem to exhaust the patient and, on the contrary, on other occasions the vomiting symptom is perfectly tolerated by the patients. In general, many attacks of intense vomiting can correspond to functional diseases, and paradoxically, moderate vomiting can be due to an organic disease with a poor prognosis.

4. Quality. This is a very important factor, since the observing clinician can make interesting semiological deductions, depending on the constituent material of the vomit. This clinical study represents a useful advance of the complementary data provided by the laboratory. Let's analyze the most common types:

a) Alimentary vomiting. The nutritional nature of vomiting frequently leads one to think of vomiting due to poor evacuation, and it is always important to simultaneously analyze the time factor, as we previously studied. If the food appears whole, that is, almost undigested, it must be due to an early postprandial reflex phenomenon, corresponding to the psychovegetative type, some gastritis or pregnancy, as the most frequent examples. If the food is already in the digestion phase, more or less attacked, it will correspond to a vomit of poor evacuation as observed in many pyloric ulcers, cholecystopathies, juxtapyloric perivisceritis, benign tumors and especially in malignant tumors of the stomach with pyloric involvement.

As a cause of food vomiting, let us also mention some processes of extreme primary gastric atony (visceral ptosis), or secondary to an asystolic stomach due to advanced obstruction.

The smell of food vomit depends on the time of gastric permanence; it can be more or less rancid or acidic (due to hyperchlorhydria or fermentation) and sometimes putrefactive in ultra-late types. The quantity, color and other organoleptic characteristics are variable and depend fundamentally on the food ingested, the residence time and the secretion material that is added. In large obstructions of the pylorus, sometimes more is vomited than eaten, due to the added gastric secretion.

b) Watery vomiting. It is a vomit that reminds us of water. The patient notices the acidity of the vomited material, the rancid or acidic smell, and the quantity is sometimes quite abundant. This type corresponds from vomiting to phenomena of hypersecretion and hyperchlorhydria (Reichmann gastrosucorrhea syndrome).

It may also correspond to a purely functional process, vagotonia, tabetics, or be secondary to hyperacid gastritis, or a gastric or duodenal ulcer, which produces vomiting with hypersecretion, acting as an irritating thorn.

c) Mucous vomiting. In this type, which is easy to identify due to its viscous, thick appearance, mucorrhoea or myxorrhea of the stomach is objective (Hayem syndrome or gastromyxorea). It corresponds perfectly to cases of chronic superficial gastritis, common in alcoholics.

d) Bilious vomiting. Bitter in taste, greenish-yellow in color and sui generis in smell. It is a type of vomiting always related to the duodenum and bile ducts, which requires a more or less intense bile reflux from the duodenum to the stomach for its production. In some cases, bilious vomiting occurs after food vomiting and in most cases on an empty stomach or on an empty stomach. Another interesting fact in the semiological interpretation of bile vomiting is that for its production the ampulla of Vater, as well as the duodenum and the pylorus, must be permeable to allow bile reflux.

Bilious vomiting is observed in duodenitis, in some types of duodenal ulcer, and in cholecystopathies.

e) Porraceous vomiting. This type depends on the presence of upper intestinal material, where mixed elements of the duodenum, the biliary tract and the initial jejunum can be identified. The smell is penetrating, slightly foul, and the color is dark green, which is reminiscent of the joint, from which its name is derived. It generally precedes fecaloid vomiting itself, and corresponds to upper intestinal occlusions.

f) Fecaloid vomiting. Its content is frankly fecal, with the corresponding organoleptic characteristics (smell, flavor, etc.). It is observed in lower intestinal obstructions. It can also occur in cases of gastrocolic fistulas.

g) Vomiting of foreign bodies. Although it is rare, we must keep in mind the possibility of identifying in this type of vomit: parasites; particularly fragments of animal, vegetable or mineral substances previously ingested (bezoariasis), which form true intragastric tumors.

h) Bloody vomiting. It will be included for study in the chapter on hematemesis.

5. Start mode. We can distinguish spontaneous vomiting clinically, as occurs in most cases in which this symptom is established in this way. Peripheral or reflexogenic vomiting is almost always spontaneous and central vomiting always is.

We also distinguish induced vomiting, in which the patient puts his finger down his throat or drinks hot bicarbonated water or some emetic. Induced vomiting is always peripheral and is observed in many cases of hyposthenic gastric dyskinesia, with a sensation of great retention of food (on the part of the patient), which forces him to induce vomiting.

6. Way to calm down. This factor is closely related to the intensity factor that we previously studied.

Sometimes vomiting calms easily when the gastric contents are emptied or the determining reflex action ceases. Other times it is difficult to calm them, which does not necessarily imply that there is organicity or seriousness of the illness that causes them.

7. Associated symptoms. Vomiting associated with:

a) Epigastric pain and hematemesis. It falls within the semiography of the symptomatic triad cited so often: complicated ulcers, tumors and other organic gastropathies.

b) Pain only, which sometimes calms. It makes one think of pyloric ulcers, cholecystopathies, appendicitis, among others.

c) Symptoms of other systems. It makes one think of the corresponding extradigestive diseases: with nephritic colic in renal lithiasis; with cough, in pulmonary tuberculosis; with dyspnea, in heart failure; with plexalgia and neurological symptoms, in tabes; with migraine, in hepatoallergy; with hypertension and oliguria, in azotemic nephritis; etc.-tera.

d) Rich symptomatic procession of psychovegetative symptoms. It requires the search for neurovegetative dystonias, psychoneuroses, psychopathies and endocrinopathies. They are patients who, in parallel with vomiting, present palpitations, sweating, hot flashes, crying, nervous excitement and other symptoms of this type.

e) Headache, constipation and projectile or spindle vomiting. It forces us to think about diseases that cause central vomiting due to intracranial hypertension; especially brain tumors.

Finally, the analysis of the associated symptoms will help a lot to know the cause of Vomiting: other factors such as exogenous toxins (emetine, etc.) or endogenous must also be taken into account.

8. Course. In this section we summarize the assessment of all the factors previously studied:

a) A regular, defined course of vomiting with precise characteristics facilitates the orientation of the diagnosis, which generally corresponds to processes of organic

b) On the contrary, vomiting with an irregular, whimsical or variable course suggests functional processes generally of psychovegetative cause.

HEMATEMESIS

It is a symptom of great importance within the gastric triad, particularly because of the fear it instills in patients and their families, because as an old Spanish saying goes, blood comes from the mouth, neither too much nor too little.

Concept

It is defined as the expulsion of blood through the mouth, with all the characteristics of vomiting; that is, preceded by nausea. It's a real vomit of blood.

Differential diagnosis

It is necessary to consider that not all expulsion of blood through the mouth is hematemesis, which is why it is appropriate to differentiate it from:

1. Stomatorrhagia or hemorrhage produced in the oral cavity itself. A quick physical examination of the mouth will be enough to verify a stomarrhea and locate its origin.

2. Epistaxis, in this case the bleeding comes from the nasal passages and can be expelled through the mouth.

3. Hemoptysis, a purely respiratory symptom, which consists of the expulsion of blood through the mouth, accompanied by cough and retrosternal tickling. The blood expelled is foamy, aerated, shiny and is accompanied by respiratory symptoms, both before the hemorrhage and after.

In hematemesis, blood expelled as vomit may be accompanied by gastric juice or food remains; It is red or black and is not foamy.

On some occasions, blood coming from the nose, mouth, or respiratory tract is swallowed consciously or unconsciously to be later vomited, then it is called secondary hematemesis.

Among secondary hematemesis we must consider those self-induced by psychopaths and for simulation purposes. These subjects may secretly bleed and swallow the blood and then have hematemesis.

Finally, let us point out false hematemesis, in which cases a material that can be confused with blood is vomited, for example, beets, currants, red wine, sausages, strawberries, etc.

Semiogenesis or pathophysiology

Hematemesis, like any other hemorrhage, has a predominant anatomopathological pathogenic substrate, although it can also be caused by a pathophysiological alteration. Let us point out as fundamental semiogenesis the lesions of the vessels: arteries, veins and capillaries, which allow the outflow of blood with greater or lesser intensity. The most common vascular lesions are: rupture, thrombosis, embolism, arterial and venous exulcerations, as well as other parietal alterations of the vessel: softening, sclerosis, aneurysms, etc. It is also important to remember the neoformation of vessels (angiomas).

On other occasions, due to functional alterations, changes in intravascular pressure are established, such as arterial vasodilation and venous stagnation, determined by various local or general causes that facilitate the rupture or dilaceration of its wall and, therefore, hemorrhage. Finally, bleeding may occur due to alteration of any of the factors involved in blood clotting, which constitutes the pathological hemorrhagic syndrome of blood dyscrasias.

It is important to note that for hematemesis to occur, the presence of a certain amount of blood produced quickly must be associated with the motor phenomena of vomiting caused by irritation of the mucosa. It is evident that if, on the contrary, the bleeding is discreet and slow, the spilled blood can pass into the intestine, be digested and come out through the anus in the form of a mane, a symptom that we will study in the chapter on intestinal symptoms. There may be, and this is frequent, both symptoms, hematemesis and melena.

Semiography and semidiagnosis

We will analyze the following factors:

1. Intensity. According to the amount of blood expelled, we can classify hematemesis as intense, medium and light.
   a)) Intense or massive. It is generally due to an ulcer due to the pathophysiological circumstances already explained (vessel injuries). It can also be due to rupture of esophageal varices secondary to portal hypertension, toxic or drug-induced erosive gastritis and, as an exception, rupture of a sclerotic or aneurysmal vessel.
   b) Medium intensity. They are hematemesis from gastritis, for example.
   c) Light intensity. This type of hematemesis, paradoxically, is sometimes observed in some malignant tumors, since benign tumors generally bleed more abundantly. We will remember with Dieulafoy that gastric cancer almost never produces large hematemesis.

2. Quality. Red and black hematemesis are distinguished:
   a) The red ones are linked to the sparkling character of the vomited blood, a sudden phenomenon generally dependent on the massiveness of the bleeding.
   b) The black ones are more discreet or slower in their production, which is why they allow the previous attack of gastric juice.

   Red hematemesis is observed more in ulcers, and black hematemesis in gastritis, some neoplasms, etc., but this must be considered in relative terms.

3. Associated symptoms. It is an important factor, as it considerably guides the anamnestic type diagnosis. Thus, when hematemesis is associated with:
   a) Epigastric pain, rhythmic periodic, calmed with food. Strongly suggests the diagnosis of an ulcer.
   b) Alimentary vomiting. It suggests a possible juxtapyloric injury.
   c) History of alcohol or jaundice, intestinal disorders and flatulence. Suggests liver diseases.
   d) Atypical epigastric pain, dyspeptic symptoms and weight loss, in a previously healthy subject. It suggests cancer.
   e) Easy bleeding of the gums, bruises or purple spots on the skin. Suggests blood clotting disorders.
   f) On the contrary, sometimes hematemesis surprises a patient, with practically no history or other associated symptoms. This type is known as silent hematemesis, and it can correspond to an acute onset ulcer, a diaphragmatic hernia, a pyloric prolapse or a portal hypertension syndrome. Benign tumors and gastroduodenitis sometimes also produce silent hematemesis.

SMALL SYMPTOMS 0 DYSKINETIC SYMPTOMS

At the beginning of this chapter we made some general considerations in the overall study of the so-called small dyspeptic or dyskinetic gastric symptoms and also in the classic criteria that grouped them into hypersthenic and hyposthenic according to Robin's classification.

We will group them according to the classic criterion considering that it offers certain advantages from the didactic point of view.

HYPERESTHENIC DYSKINETIC SYMPTOMS

They are:

-Aerophagia and aerophagic belching.

- Gastric regurgitation.

Non-aerophagic belching.

- Gastric burning.

-Hiccup.

Let us point out, however, that sometimes these symptoms may appear associated with hyposthenic phenomena.

Aerophagia and aerophagic belching

Concept

This is understood as the exaggerated swallowing of atmospheric air, more or less silently (aerophagia), followed by the expulsion of air, in a noisy manner (aerophagic belching).

As we can see, pathological aerophagia almost always produces belching, but not all belching is air, there is also the expulsion of other gases, which we will study under the title of non-aerophagic belching.

We must accept occasional aerophagia and aerophagic belching, which is performed at a very moderate degree of intensity, usually after finishing eating. It is a relatively physiological phenomenon, which in the social order must be considered rude, although in past times it was a sign of good taste in a complimenting guest, as proof of complacency towards the host. Let us also mention the aerophagia and physiological aerophagic belching of the infant. Therefore, we can consider this symptom as the exaggeration of a physiological or normal phenomenon that causes obvious discomfort to patients who suffer from it.

Patients speak synonymously, that they 'belch'" or "burp", that they "expel gas", that they "repeat", that they "return", etc. Sometimes identification is facilitated When the doctor hears the intense noises that patients produce when expelling air, they do this with great satisfaction in front of the doctor to objectively demonstrate their condition.

Semiogenesis or pathophysiology (fig. 57.16)

As we have advanced in the concept, it is evident that in the phenomenon of swallowing air with a pathological intensity, the excess air is imprisoned, either in the lower portion of the esophagus, or in the air chamber of the stomach, to be expelled immediately afterwards.
Kantor points out the terminal portion of the esophagus as the appropriate site for the accumulation of air that the patient swallows and which is belched by retroperistalsis, and the repetition of the phenomenon.

Álvarez speaks of retroperistalsis or reversal peristalsis from the cardia. Swallowed air also accumulates in the stomach chamber. It is assumed that the physiological reflex that controls the entry and exit of atmospheric air in certain circumstances originates in this area, particularly postprandial gastric filling and after ingestion of bicarbonated drinks or soft drinks.

This pathological phenomenon may be due to several fundamental factors together: increased juxtacardial accumulated air pressure; Primary vagal hyperreflexia and hyperreflexia secondary to an altered gastric mucosa acting as an irritating thorn. From any of these phenomena, or from the concurrence of several at the same time, retroperistalsis is established and the symptom originates.

It can also occur in hypotonic stomachs with poor evacuation, as a compensation mechanism to promote gastric emptying.

Sialorrhea, which causes continuous swallowing with air entrainment, markedly favors the production of the symptom we study.

semiography

It is not possible to carry out a detailed study of the semiological value of aerophagy and aerophagic eructation due to their particular characteristics; however, their postprandial character can be noted, earlier than later; its noisy, sonorous, intense character; as well as its association with other symptoms, especially gastric fullness and gastric regurgitation.

Semidiagnosis

The diseases that most frequently give rise to this symptom are those that cause sialorrhea, for example, pathological processes of the mouth and nasopharynx (stomatitis, nasopharyngitis, dental conditions).

With preference we must mention the aerophagia of psychoneurotics, anxious people and patients with vagal hypertonia, in whom it is established

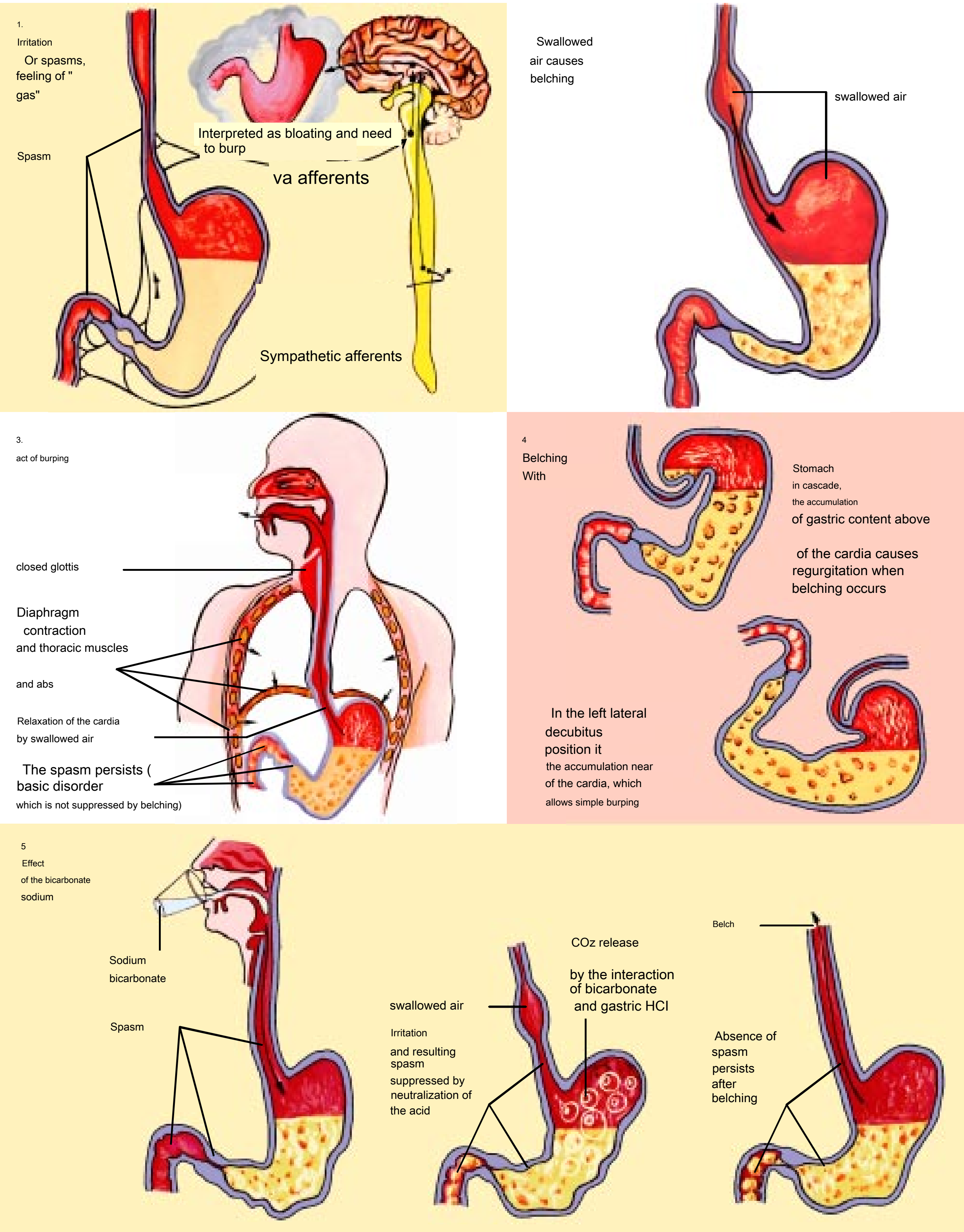

1.
Irritation
Or spasms, feeling of " gas"
Spasm
Interpreted as bloating and need to burp
va afferents
Sympathetic afferents
Swallowed air causes belching
swallowed air
3.
act of burping
closed glottis
Diaphragm contraction and thoracic muscles
and abs
Relaxation of the cardia by swallowed air
The spasm persists (basic disorder
which is not suppressed by belching)
4
Belching With
Stomach in cascade, the accumulation of gastric content above
of the cardia causes regurgitation when belching occurs
In the left lateral decubitus position it
the accumulation near of the cardia, which allows simple burping
5
Effect of the bicarbonate sodium
Sodium bicarbonate
Spasm
swallowed air
Irritation and resulting spasm suppressed by neutralization of the acid
COz release
by the interaction of bicarbonate and gastric HCl
Belch
Absence of spasm persists after belching

Fig. 57.16 Semiogenesis of aerophagy.

a vicious circle of aerophagia and belching of air, resistant to all treatment. These patients intensify the maneuvers to swallow air, which causes gastric fullness, the symptom of which they try to get rid of by swallowing more air. They reproduce their symptom before the doctor or their concerned relatives, to demonstrate the seriousness of their illness. Psychiatric therapy is necessary in these cases.

Let us also mention among the diseases that cause this symptom, upper parcel gastritis, lower esophagitis, hiatal hernia and other injuries of the juxtacardial region.

gastric regurgitation

Concept

It is defined as the expulsion, from the stomach towards the mouth, of small amounts of gastric contents. Generally, gastric regurgitation is an early postprandial symptom and is closely associated with aerophagia and aerophagic belching, since in a pathophysiological order it should be considered as the same phenomenon but more exaggerated; that is, produced with greater intensity. It is identified because the patient reports that the food or gastric juice rises to the mouth, as in "buchados", which constitutes its synonymy.

Gastric regurgitation is not accompanied by nausea, differentiating it from vomiting, from a clinical point of view.

Semiogenesis or pathophysiology

In gastric regurgitation, antiperistalsis is established from the upper parts of the stomach and it is possible that the same phenomenon exists simultaneously in the terminal part of the esophagus (esophageal regurgitation, a symptom already studied). It is a phenomenon of intrinsic muscle contracture, in which the extragastric muscles intervene little or not at all, a fact that clearly distinguishes it from vomiting. Perhaps the greater or lesser competence of the cardia plays its role.

Generally, antiperistalsis of regurgitation corresponds to simple functional alterations, but it can also occur due to organic lesions of the juxtacardial region. The regurgitated material is food or gastric juice, always in small and relatively repeated quantities. In well-fed infants, gastric regurgitation is quite common, and the pediatric phrase " the child who vomits becomes fat" is known, which, when corrected, should be "the child who regurgitates becomes fat," which in these cases depends on excess ingestion.

semiography

There are some clinical details to study in gastric regurgitation. The character of the regurgitated material is variable, it can have a different taste and smell: sour, bitter, fetid, which depends on the regurgitated material, whether it is gastric juice, bile or late-digested foods.

Semidiagnosis

The diseases previously mentioned as producing aerophagia and aerophagic eructation can all also be considered in the semiological significance of gastric regurgitation. Juxtacardial lesions such as diaphragmatic hernia, lower esophagitis, upper gastritis, some types of gastric ulcers, and particularly duodenal ulcers with marked hypersecretion and gastric retention, should preferably be noted. Gastric regurgitation may also be observed in some cases of functional or organic hypotony or atony with or without pylorus obstruction.

Non-aerophagic belching

Concept

It is defined as the expulsion of foreign gases into the atmospheric air, with similar characteristics to aerophagic eructation. Its identification is perhaps linked to the different smell and taste of said belches, which patients call belches of rancid or fetid gases.

Semiogenesis or pathophysiology

In this symptom, as its name indicates, what is expelled are gases of fermentation or putrefaction accumulated in the stomach and secondary to more or less marked gastric retention. This is frequently observed in cases of prolonged pyloric obstruction, in which the stomach loses its peristalsis and tonus, until reaching what is called gastric asystole, with stagnation of food for long periods of time.

The composition of these gases is variable, carbon dioxide (from carbohydrates) and sulfurized hydrogen and ammonia (from proteins).

Sometimes, gases are not generated in the stomach, but in the proximal intestine and reflux to the stomach to be belched.

Semidiagnosis

We can mention as diseases capable of producing non-aerophagic belching, juxtapyloric ulcers complicated with obstruction, benign and especially malignant obstructive tumors, as well as any other necessarily organic process that determines

Prolonged obstruction of the pylorus with food stagnation, such as some perivisceritis, etc. In parasitic duodenitis due to Giardia laInblia, it is common for patients to report this symptom.

Gastric burning

Concept

It is defined as the burning or burning sensation that some patients experience in their epigastric region without precisely reaching the sensation of pain.

This sensation is identified only at the level of the stomach and does not travel in any other direction; It should not be confused with heartburn. It can be observed after meals, early or late. Patients speak of burning in the stomach", of a sensation like "vinegar in the stomach".

Semiogenesis or pathophysiology

Gastric burning is due to the contact of hyperacid gastric juice with an altered, more or less inflamed gastric mucosa. Let us say immediately that a normal mucosa resists the contact of mechanical agents (rods, other foreign bodies, etc.), physical agents (heat, cold) and chemical agents (hydrochloric acid), without the human subject perceiving an unpleasant sensation, which is why we speak in physiological terms of the insensitivity of the gastric mucosa. Alcohol, in a certain concentration, is pointed out as the exception to this postulate.

In pathological conditions, that is, when the mucosa is inflamed, all contact is very sensitive and causes this symptom, which in intense cases is accompanied by burning or burning pain, which we study in the section dedicated to the quality of epigastric pain.

As we have said, the most important factor is hyperchlorhydria, although the possibility of excess fermentation and putrefaction acids occurring in exceptional cases of gross food retention in the stomach is also noted. The psychic factor of perception is important in the genesis and intensity of this symptom, without great alteration of the mucosa, in cases of marked hyperchlorhydria in hypersensitive subjects.

tives.

There may also be gastric burning, with low acidity levels and even anachlorhydria, as also occurs with heartburn, in which case the phenomenon is rather motor and produces an equivalent subjective sensation.

Semiography and semidiagnosis

As a disease that typically produces gastric burning, let us point out gastritis, especially the chronic and superficial type, in which case the pathogenic circumstances are ideal, especially if accompanied by hypersecretion and hyperchlorhydria. Local allergy is an important factor.

Heartburn can be early postprandial, late postprandial, or also occur on an empty stomach.

In cases of early postprandial time, severe gastritis should be considered, even if at that time there are not yet high acidity levels.

The late postprandial appearance of heartburn suggests gastritis, but in this case the acidity, already high, is a determining factor.

In cases of gastric burning on an empty stomach, permanent hypersecretion syndromes associated with gastritis are considered.

As an exception, gastric burning can be observed in cases of pyloric obstruction accompanied by acidity from fermentation or putrefaction: ulcers or stenosing tumors of the pylorus.

Hiccup

Concept

It is defined as the annoying, sometimes unbearable, sensation due to clonic contractions of the diaphragm, of greater or lesser intensity. It is known as a synonymous term, singlet, and its identification is very easy for the patient and the doctor, due to its obvious objectification (sudden movement of the upper abdomen).

Semiogenesis or pathophysiology

In its intimate mechanism, the more or less rhythmic diaphragmatic contraction with closure of the glottis should be noted.

It is a respiratory reflex due to the clonic contraction of the diaphragm, which causes a sudden inspiration, causing air to pass through a narrowed, almost closed glottic opening, producing a characteristic noise. When the diaphragmatic contractions occur (single or in bursts) the attack or crisis of hiccups or singlet occurs.

This is a type of reflex very similar to vomiting. In this reflex arc there are afferent or centripetal pathways, made up of the pneumogastric, sympathetic and phrenic nerves. The centers are found in the medulla, neighboring the emetic centers, and the efferent or centrifugal pathways are made up of the phrenic pathways, which innervate the diaphragm.

The reflex can arise from any place, but it is produced more easily by following the law of the distance of the irritating thorns stated so many times, from the diaphragm and its vicinity. Vagal and phrenic irritation are important factors.

A retrospective look at this book will confirm the pathogenic similarity of hiccups and vomiting. Has to peripheral hiccups and central type hiccups can be distinguished. In peripheral hiccups, as we have explained, the mechanism will be reflex, from any irritating spine in the diaphragmatic vicinity: diaphragm, upper stomach, great vessels at the diaphragmatic crossing, lower esophagus, etc.

Central hiccups will be due to direct stimulation of the bulbar centers, at the expense of any blood alteration or changes in cerebrospinal fluid pressure.

Semiography and semidiagnosis

We can mention as diseases capable of presenting hiccups by reflex mechanism: lesions near the diaphragm, liver disease and splenopathy with an increase in the size of these organs, diaphragmatic hernias, lower esophageal lesions (ulcerations, inflammations, etc.), gastritis, gastric ulcer, stomach cancer and some pancreatitis. Lesions of the heart, pericardium and pleura, phrenic neuritis, large goiters, as well as aortitis and aortic aneurysms also cause hiccups due to irritation of the phrenic.

Less commonly, hiccups are seen in some conditions more distant from the diaphragm: peritonitis, for example.

On the other hand, at the expense of the central mechanism, many septicemias and toxemias in which hiccups are a sign of poor prognosis can be cited as causes of hiccups, as well as central hiccups in brain tumors and encephalitis.

In hypersensitive people, an attack of hiccups or hiccups can be triggered due to external contact with cold water or drinking very cold or hot liquids, also when they suffer from emotions, situations that cause psychic hiccups and that explain the many maneuvers, sometimes picturesque, that the masses use to stop a hiccup attack, with the aim of diverting the patient's attention or causing a stronger impression.

HYPOSTHENIC DYSKINETIC SYMPTOMS

In this group we have:

- Gastric fullness or fullness, heaviness and satiety.
- Nausea.

Epigastric jump.

Gastric fullness or fullness, heaviness and satiety

These subjective symptoms are explained as a whole.

Concept

Let us define these symptoms as the sensations that many stomach patients experience and which consist of a great occupation of the organ, that is, the sensation of being very full, as if they had eaten a lot; Patients loosen their girdles or belts to relieve the sensation of epigastric pressure. In other cases, they talk about their stomach being "heavy" in the epigastric region, with a feeling of distention, as if it had acquired a large volume. They also report a feeling of satiety, that is, feeling full as soon as they start eating as if they were easily satisfied. There is a lack of proportion between fullness and the actual amount of food ingested. These symptoms, always postprandial, can be early or more or less late and of variable duration.

Semiogenesis or pathophysiology

The pathophysiological basis of this group of symptoms is eminently functional and of a motor or dyskinetic nature, although as an immediate functional consequence they can also be observed in organic processes that affect gastric capacity: neoformations and chronic inflammation, for example.

They are most frequently established as a consequence of phenomena of hypotonia, hypoperistalsis and hypomotility, which may or may not coincide with hyposecretion or hypochlorhydria.

Semiography and semidiagnosis

Compared to fullness, fullness, etc., we should preferably think about gastric ptosis with hypotonia, typical of subjects with Stiller's asthenic habit.

Let us also point out the fullness or fullness that some cases of gastric cancer and gastritis present.

Nausea

This symptom is considered by many authors to be hyposthenic, although it can sometimes be hypersthenic.

Concept

Although we studied nausea as a precursor to peripheral vomiting, it also deserves to be studied as an isolated symptom. It is defined as the vague, unpleasant, repulsive and depressive sensation that many patients experience in their epigastric region. Patients identify their nausea with the sensation of "sinking or "depression in their stomach" with the desire to vomit, even if the vomiting itself is not performed. Frequently, this sensation is reported by extending the throat, with phenomena of weakness.

faintness, of emptiness, 'as if one's life were passing away'. Sometimes nausea is associated with circulatory alterations, hot flashes, palpitations, dizziness, sweating, etc. Patients use the terms synonymously: retching, fatigue, urge to throw, etc.

Semiogenesis or pathophysiology

Alvarez points out a mechanism of nausea

antiperistalsis. Its pathophysiology is similar to that of peripheral vomiting, with reflexes of lower intensity. Antiperistalsis from the duodenum is pointed out by Keeton. Ryle similarly points out hyperkinesia and hypokinesia in the mechanism of nausea production. It is noted in cases of hypersecretion and hypertonia, as well as in cases of obvious hypotonia with an almost total absence of peristalsis, as Barcley verified under the fluoroscopic screen in a case of migraine. A certain central action can also be noted, as occurs with nausea associated with vertigo, in motion sickness.

Finally, the sensory and psychic factor also intervenes in its production. Many people smell something unpleasant, see certain things or think about certain spoiled foods or waste and feel nauseated, using the well-known phrase that their stomach turns or turns upside down. This symptom is more frequent in functional processes than in organic ones.

Semiography and semidiagnosis

Let us point out the nausea of gastritis, especially alcoholic ones, which occur on an empty stomach. Let us also point out carcinomas of the stomach, cholecystopathies, jaundice, both obstructive and hepatocellular, and chronic appendicitis, as preferred examples. Gordon points out the presence of the symptom of nausea in 28 of 37 biliary patients and, on the contrary, only in 34 of 131 patients with gastric or duodenal ulcer.

Nausea occurs in patients with chronic pharyngitis and in pure dyskinetics with gastric ptosis and solaritis (irritation of the solar plexus); in circulatory and kidney diseases in the decompensation phase, in many tuberculosis patients and in the majority of cases of meningitis and brain tumors. Nausea is very important for patients with headaches, especially migraine-type headaches.

Because this symptom is so common, the assessment of the associated symptoms is very important in guiding the diagnosis. For example, with vertigo, you think in labyrinthine processes; with headache, in migraine; with hypertension, in nephritis, etc. We must also point out the nausea of pregnancy.

epigastric jump

Concept

It is defined as the jumping or throbbing sensation that patients experience in the pit of the stomach or epigastric region. They identify this symptom as an annoying, unpleasant sensation, as if their heart was beating in their stomach. They use as synonyms the terms: "padrejón or mal de padre", "madrejón or mal de madre", widely used among peasants, who try to get rid of the illness ("empacho") through massage of the region, performed by people who are supposed to have "spiritual virtues" for its realization. With the cultural development achieved in our social system, by taking education and medical care to the mountains, this practice is used less and less every day.

Epigastric skip is simply a subjective symptom of a neurovegetative nature, which we study here due to its close relationship and association with small gastric dyskinetic symptoms or symptoms.

Semiogenesis or pathophysiology

It consists of erethism or excitability of the abdominal aortic segment, which is reported by the patient with greater intensity at the level of the epigastrium. Epigastric leap is supposed to be associated with sympathetic hypertonia, with irritability of the solar plexus. It generally coincides with gastric ptosis and a good number of other dyskinetic symptoms, predominantly of the hyposthenic type.

Semiography and semidiagnosis

It is observed in digestive ptosis with or without gastric hypotonia, in solaritis, in patients with neurovegetative dystonia with sympathetic predominance (sympathicotonics), in patients with psychological instability, in patients with anxiety or anxiety psychoneurosis, etc.

We must point out that there is also an epigastric jump due to lesions of the aorta: sclerous aortitis or aortic aneurysm, whose processes can also have this subjective component, but it is easy to make the differential diagnosis by palpating the aorta, which is in its normal structure, which is why this symptom is also called aortism.

DIGESTIVE SYSTEM. SEGMENT SYMPTOMS

ENTEROCOLICORRECTAL

58

Introduction

The symptoms corresponding to this segment are:

Great symptoms or capital symptoms	Enterorrhagia and melena Constipation Diarrhea
Small symptoms or dyskinetic symptoms	Abdominal distension or bloating borborygmi Expulsion of gas through the anus Colic heartburn
Particular symptoms of the rectum and anus	Rectal pain or proctalgia Rectorrhagia rectal discharge anal itching Rectal heaviness and protrusion Anal burning

This is an extensive group of symptoms with which we must become familiar and which, in general, will guide us towards the diagnosis of diseases of the small intestine, colon, rectum and anus. Once again, let us remember that this orientation is not absolute, since we can find some of these symptoms, particularly non-epigastric abdominal pain, as corresponding to some disease outside the intestine and even the digestive system. The abdomen, fundamentally digestive, contains organs from other systems: urinary, genital, circulatory, endocrine, nervous, etc.

Just as we did in the group of gastroduodenohepatobiliopancreatic symptoms, we have divided this group of symptoms into large and small symptoms, according to their semiological hierarchy. In general, the major symptoms or capital symptoms: non-epigastric abdominal pain, enterorrhagia and melena, constipation and diarrhea, suggest preferentially organic conditions of the intestine, although some may also correspond to functional diseases. On the other hand, in the presence of small symptoms or dyskinetic symptoms: abdominal distension, borborygmi, expulsion of gas through the anus, etc., We will guide the diagnosis of functional diseases or intestinal dyskinesias, although they may also be present in organic enteropathies.

The concurrence or association of two or more capital symptoms will bring us even closer to the possibility of significant intestinal injuries. Lower abdominal pain associated with progressive constipation and enterorhagia suggests a possible tumor of the lower portions of the intestine; tumor, on the other hand, occlusive and hemorrhagic. The presence of even one of these capital symptoms requires further semiological analysis.

The group of small symptoms as a whole has an evidently motor-producing mechanism, hence the equivalent terminology of dyskinetic symptoms. Sometimes, the intimate pathophysiological factors are complex, therefore their semiological assessment is difficult.

Finally, we will study in a separate section the subjective symptoms of the rectum and anus, which correspond particularly to the anorectal nosology, of great frequency and importance, which justifies the existence of the specialty of Coloproctology. This semiology is medical and surgical, according to the treatment methods. This group of symptoms: rectal pain, rectal bleeding, rectal discharge, anal itching, rectal heaviness and protrusion, and anal burning, most often have a more organic than functional character.

MAJOR SYMPTOMS 0 CAPITAL SYMPTOMS

The capital symptoms of the enterocollicorrectal segment

Are:

- Enterorrhagia and melena.
-Constipation.
Diarrhea.

ENTERORRAGIA AND MELENA

Concept

Enterorrhagia can be defined as the expulsion of fresh blood from the anus. In enterorrhagia, the blood shed throughout the intestine, for any reason, is expelled to the outside, quickly, without fundamental modifications. It is identified, therefore, as bright, red blood, like that which could flow from a wound. The common people use the term "fine blood" as a synonym.

The melena is the expulsion of digested blood through the anus. The blood is actually defecated, mixed evenly with the feces, as something homogeneous; It offers a creamy, shiny, tarry black appearance. It is easily identified as previously digested blood, expelled more slowly, under the effects of normal peristalsis. The synonym used is " black blood like chapapote."

In enterorrhagia, associated lipothymia is more common than in melena, although it all depends on the amount of blood lost. On the contrary, small amounts can be expelled and sometimes the bleeding is so small that neither enterorrhagia nor melena are determined, which is why the phenomenon is only verified by the chemical examination of the feces.

Let us mention, of course, the possibility of non-externalized digestive bleeding (neither hematemesis, nor enterorrhagia, nor melena), which can end the patient's life.

The differential diagnosis of enterorrhagia practically does not exist, but in discrete melena the possible confusion with blackish stools secondary to the ingestion of red wine, foods rich in blood (black pudding, etc.), as well as the administration of medications that darken the stools, such as: bismuth, iron, charcoal, ratany, must be taken into account; and, finally, excess bile (false mane).

Semiogenesis or pathophysiology

In enterorrhagia, in general, it is assumed that the hemorrhagic focus is lower, close to the anus (rectum and lower colon), but the speed of exit or degree of hypermotility and the amount of blood must be assessed in all cases. Therefore, there may be an enterorrhagia due to a high injury, whenever there is hypermotility associated with a large amount of blood. In this case, there is no retention of the spilled blood and therefore, it is not modified, leaving without appreciable alterations.

In the mane, on the contrary, the hemorrhagic focus is generally high, at a greater distance from the anus (upper colon, small intestine, stomach and even esophagus), for which reason there is an obvious retention of spilled blood. This is digested and incorporated into the feces as if the patient had eaten a similar food (black pudding, for example) and the blood comes out (with normal motility), completely modified, digested, with obligatory putrefaction and therefore fetid phenomena. A minimal amount of spilled blood is needed to produce a conspicuous mane; This amount is set at around 60 mL. When produced in larger quantities, if the bleeding rate is discrete, motility is not markedly altered.

These considerations are interesting, since in the semiological assessment, enterorrhagia should suggest low or high lesions with hypermotility and massive bleeding; and the mane, in high lesions, with continuous, but non-violent bleeding or as a residue of digestion of blood whose majority has been previously expelled through the mouth in the form of hematomas.

temesis, a previously well-studied symptom associated in most cases with melena.

In the intimate production of bleeding from enterorrhagia and melena, there are the same factors already mentioned in hematemesis, such as: vascular lesions, tension changes, capillary neoformation and blood dyscrasias. We consider it sterile to repeat the analysis of these pathophysiological factors. The presence of septic factors and vascular sclerosis facilitates the production of hemorrhage.

Semidiagnosis

In the same way as we pointed out for hematemesis, the diseases capable of producing enterorrhagia and melena are numerous. It is not easy to go from symptom to disease. The intestine is very long, and in its 5-6 m, hemorrhage can occur at different levels, due to the various mechanisms noted above. As we have already pointed out, certain particularities in the production of an enterorrhagia and a mane separately, it also seems pedagogical to analyze their semiological value in an independent way.

Semiological significance of enterorrhagia

Let us preferably point out the hemorrhagic lesions of the lower portions of the colon and rectum, such as: malignant tumors, benign tumors (such as polyps), hemorrhoids, anal fissures and dysenteric processes (acute amoebiasis, bacillary dysentery and idiopathic ulcerative colitis). Let us also mention, as a cause of enterorrhagia, intestinal tuberculosis, as well as intestinal intussusception in children. Mesenteric thrombosis should also be noted as a cause of enterorrhagia.

When the enterorrhagia is rectal it is called rectal bleeding, to which we will return later, in the particular study of subjective anorectal symptoms.

Semiological significance of the mane

Theoretically, any of the diseases mentioned in the previous paragraph as a cause of enterorrhagia can cause a melena, provided that the circumstances vary and the blood spills more slowly, without hyperperistalsis, starting from places further away from the anus.

Let us also say now that all the diseases mentioned in the semiological significance of hematemesis can at the same time produce melena. The association of hematemesis with melena is obvious. The semidiagnosis of hematemesis was discussed in Chapter 57.

Let us also point out, as a cause of melena, the duodenal ulcer, both of the bulb and postbulbar; hepatolienal diseases, such as cirrhosis with esophageal varices; thrombosis of the portal system; jejunitis and ileitis of different nature. Tumors of the intestine The small intestine and the right colon are important in this relationship.

CONSTIPATION

Concept

This symptom is defined as the delay in the normal and periodic evacuation of feces, as well as its decrease in quantity, with or without alteration of its quality. It is identified when the patient tells the doctor that he does not correct daily as he used to do, but rather every 2 6 3 days, or that he corrects every day, but in an amount that seems small and in turn in dry "balls." Although the transit of food, with its subsequent changes until reaching useless residue or excrement, is variable, the average time period can be taken to be 48 hours. Patients commonly use terms synonymous with constipation; difficulty moving the belly, that they are “stuck, that they do it hard, in little quantity, etc.

Constipation, unknown to the ancients, is a consequence of urban life and civilization.

This symptom is of capital importance and of unusual frequency; Kantor points out, only for the atonic or habitual form of constipation, the frequency of 45% of the patients questioned. It is clear that we are studying constipation as a symptom, therefore, its extension is great given its wide pathogenic possibilities and semiological significance. We will study from the acute surgical constipation of an intestinal occlusion due to a strangulated hernia, to the usual chronic constipation, mentioned previously.

Semiogenesis or pathophysiology

Within the multiplicity of causes and mechanisms of production of constipation as a symptom, we will list the main factors for subsequent analysis:

However, first it is necessary to do a physiological count of defecation.

Defecation is normally carried out in three successive phases (fig. 58.1):

1. The first is involuntary. The sigmoid, which is filled from the bottom up, and the fecal matter that has just reached the rectosigmoid junction is placed in its place in the extension of the rectum, then invaginates into the rectal ampulla, creating a true active prolapse.

2. The second phase is voluntary. The head of the fecal bolus descends through the rectum and presses on the sensitive mucosa, thus causing the need to defecate. The individual exerts force and creates intra-abdominal overpressure, which thus repels the fecal bolus.

3. The third period is brief, both voluntary and reflex:

The sphincter opens voluntarily and feces are evacuated.

For the conditioned reflex to take place, the child must get into the habit of voluntary daily defecation, at a fixed time. This neuromotor education is too often disturbed by the demands of social life. Defecation rarely finds its place at the right time.

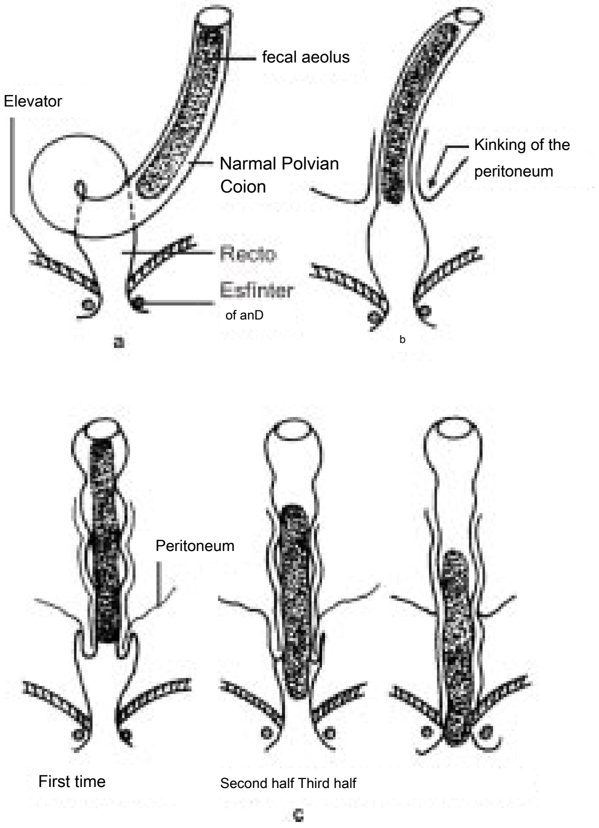
The normal sensitivity of the rectal mucosa, stimulated in vain, is eventually extinguished, as demonstrated by the introduction and inflation of a balloon into the rectal ampulla (Busson). The reflex, whose starting point is the need to evacuate, is not conditioned.

Hereditary and psychological constitutional factors (for Alexander, constipation would be a “selfish” reaction against the obligation to give), are often associated to alter the neuromotor functioning of the terminal intestine.

The most important pathophysiological factors are:

- Decreased permeability of the digestive tract (particularly the small and large intestine).

Fig. 58.1 Scheme of defecation: a, empty rectum before defecation; b, preparatory time for defecation; c, the three times of defecation itself.



-Alterations in neuromotor balance.

Alterations of the humoral agents of peristaltic regulation.

- Decrease in water intake.
- Hyperactivity of the digestive enzyme action and intestinal
- Lack of waste substances in the diet.
- Accessory factors.

Decreased permeability of the digestive tract (particularly the small and large intestines)

Anything that occludes the lumen of the intestine, at any point along its long path, whether in the small or large intestine, will be an obvious cause of constipation. This phenomenon can occur suddenly or gradually. In turn, intestinal impermeability can be determined by intrinsic occlusion or extrinsic compression. Sudden impermeability causes acute constipation, and slow or gradual, subacute or chronic constipation, both also called surgical constipation, since the diseases that produce them generally require surgical intervention for possible cure. It is a type of anatomopathological substrate constipation or organic constipation.

Neuromotor balance alterations

We have intentionally used the term neuromotor balance to mean that there is a harmonious balance of a series of elements of the neuromotor system of the intestine, which guarantee transit, as well as the normal and periodic expulsion of feces through the physiological act of defecation. Fundamentally, muscle tone, the vagosympathetic extrinsic nervous system, and the autochthonous intrinsic nervous system, all acting together in a physiological manner, ensure normal motility or transit.

Any alteration of variable intensity in one or more of the aforementioned elements may determine (if said alteration is established with a hypodynamic depressive character) the so-called neuromotor or functional constipation.

The fundamental cause of functional constipation is the alteration of the nervous mechanism that regulates the motor activity of the large intestine. It is a true dyskinesia, and can be considered a psychosomatic symptom, in which the shock organ is the colon, given that any psychological alteration can lead to it. This fact can be explained through the discharge of catecholamines, provoked by tension, which inhibits the peristaltic activity of the colon and causes contracture of the sigmoid, which would therefore act as a sphincter area.

Almy verified the influence that emotions have on intestinal motility through rectosigmoidoscopy. Hostility, anger and, in general, negative emotions determine segmental, non-propulsive contractions, which lead to constipation or habitual constipation. On the other hand, in the individual without tension the mucosa remains normal and the intestine is permeable.

This constipation has a pathophysiological substrate.

In other circumstances in which the alteration is oriented towards excitement and hyperdynamia, the opposite symptom will be observed, diarrhea, which we will study later.

Alterations of the humoral agents of peristaltic regulation

In peristaltic regulation there is a whole series of circulating agents in the blood whose action is parallel to the mediating substances that are an integral part of the intestinal cholinergic and adrenergic systems. The decrease in stimulant or peristaltin type substances, or the increase in other depressive and inhibitory type substances, can cause the symptom of constipation.

Let's mention among the peristaltins (which can decrease): histamine, acetylcholine, pilocarpine, esterine, Na and K ions, vitamin B, oxytocin and, finally, bile salts and among depressant substances (which can increase): adrenaline, atropine, morphine, Cay Mg ions, an alkaline pH, as well as some amines and phenols due to intestinal putrefactions.

Hormones from the digestive tract, which we will study later, can regulate intestinal motility; Some of them, such as cholecystokinin, pancreocymin and the so-called motilin, activate this motility.

Alterations in these substances can be due to multiple causes, both endogenous and exogenous.

Decreased water intake

The production of the constipation symptom is evident, due to lack of water or liquid ingestion. Dry diets are constipating or constipation-producing.

Hyperactivity of digestive enzyme action and intestinal hyperabsorption

In certain rather physiological circumstances, by compensation, such as convalescence from illnesses or following fasting phases, this constipation mechanism is established.

Lack of waste substances in the diet

A minimum of residual substances in food is normally needed; These substances are not absorbed and contribute to physiological bacterial life; In addition, they absorb water, fluidize the feces and act as peristalsis promoters, generating gases and providing a certain level of acidity to the environment. They are represented by cellulose and lignin. By decreasing the diet, it becomes constipating. This factor serves as a basis for making the best therapeutic deductions, both dietary and medicinal.

Accessory factors

Let us call this another group of factors that are still of relevant importance in what is commonly called hygiene and good defecation habits.

Vicious positions for defecating, the lack of a regular and fixed schedule, the difficulties of adequate premises and environments, and the abuse of laxatives that continually disturb intestinal motility, are factors that cause constipation.

semiography

Let's look at the following factors, listed in Table 58.1:

Table 58.1

Semiographic characteristics of constipation
1. Intensity 2. Quality 3. Start mode 4. Way to win 5. Associated symptoms 6. Course.

1. Intensity. There are two types of constipation: very intense, in which the patient can go for around seven days without spontaneously moving the belly, and less intense, in which the patient moves it more frequently. The amount of excrement in the evacuation must be taken into account, since it is possible that the belly moves every 24 hours, but in a small amount, sometimes insignificant. Surgical type constipations are generally much more intense than medical type constipations. An intestinal obstruction due to a tumor is more intense than usual constipation.

2. Quality. The characteristics of the bowel movement of a constipated person, reported by the patient himself, guide this semiographic analysis.

In the atonic type and rectal dyschesia, constipation describes its bowel movements as masses or balls of very dry fecal material; either constituting a single unit or several agglutinated units, all more or less spherical in shape. Other times, the patient refers to isolated pellets or pills (escíbalos) that remind us of goat-type excrement, which corresponds to the fragmentation of the excrement in the sigmoid or even in the lower portion of the descending colon, where feces stagnate, stopped by an underlying inferior spasm.

Tapered stools are also sometimes seen in cases of constipation due to terminal anal spasm (essential or due to fissure, hemorrhoids, etc.), or due to organic stricture due to rectal tumors.

3. Start mode. Surgical constipation sets in violently in acute cases, as occurs in hernia strangulation and volvulus, among others. In chronic cases (tumours) its establishment is possible more slowly and, curiously, it can be intermittent, since for a certain time it is possible that the added spastic factor is what determines the constipation. Medical constipation also has a slow and progressive onset, which is due to a neuromotor mechanism.

4. Way to overcome. An acute or chronic surgical constipation does not subside until the cause that determines it is resolved, generally through an appropriate surgical intervention. In medical cases, sometimes constipation is easily overcome only with the reestablishment of hygienic and dietary measures.

   Other times it resists much more and the use of laxatives, purgatives and enemas becomes necessary due to its intensity. It is clear that this creates a vicious circle, making said constipation increasingly more resistant, so we must carry out careful pathophysiological reasoning in each case.

5. Associated symptoms. This is a characterological factor of the greatest importance and which has led to the clinical classification of this symptom into left constipation and right constipation. It is called left constipation, which is related to the alteration of the rectosigmoid, particularly with the so-called rectal dyschesia and in which it is assumed that there are no associated symptoms. In right constipation, related to so-called colonic and cecal stasis, the presence of a symptomatic process characterized by headaches, dizziness, vasomotor symptoms, asthenia, anorexia, coated tongue, etc., has always been noted, all encompassed under the heading of intestinal autointoxication, ardently defended by many authors; But it has also been proven how mechanical distention of the rectum, a phenomenon similar to that which occurs in rectal dyschesia, is capable of generating subjective symptoms very similar to those of this symptomatic courtship. The symptoms associated with constipation are listed in Table 58. 2. Another important associated symptom is pain, in which cases the semiotician focuses on constipation of a surgical type and of a spastic nature, since the inhibitory type with hypotonia or atony is generally painless. The association with diarrhea implies the probability of concomitant neuromotor alterations, due to hypodynamia or hyper-dynamia, that is, alterations of both adrenergic and cholinergic mechanisms, which is observed in irritable, unstable colon constipation or colonic dyskinesia (neurogenic colon). There is also the concomitance of constipation with false diarrhea, consisting of the expulsion due to constipation of small and repeated quantities of a thin, sometimes bloody liquid content, which is nothing more than the product of irritation of the rectal mucosa next to the impact, commonly produced by a fecaloma or a tumor. This false diarrhea is due in its description to Mathieu, of the French school. Constipation associated with the expulsion of fresh blood or rectal bleeding leads one to think of low tumors as the cause of constipation. The predominance of psychoneurovegetative symptoms leads to neural constipation in an altered psychosomatic background. The presence associated with psychological symptoms of a depressive nature (sadness, affliction, anguish, moral depression, etc.) will guide towards the diagnosis of constipation of psychological origin, determined by a psychosis or by a depressive or anxiety psychoneurosis.

Table 58.2

Main symptoms associated with constipation
1. In right constipation or rectal dyschesia: headaches, dizziness, asthenia, anorexia, vasomotor symptoms, coated tongue 2. Pain 3. Alternating diarrhea or association of false diarrhea. 4. Rectal bleeding 5. Neurovegetative symptoms 6. Psychological symptoms: sadness, grief, moral depression, anguish, etc.

6. Course. Accidental, temporary constipation must be due to types related to dietary transgressions, defecatory hygiene, etc., while constipation with a regular, permanent course always poses chronic, neuromotor constipation, which requires a more difficult analysis. The course of acute surgical constipation is obvious due to the associated symptoms.

Semiodiagnosis (fig. 58.2)

There are multiple diseases capable of producing this symptom, as deduced from their numerous pathophysiological mechanisms. Let's list the main ones.

The symptom of organic or surgical constipation occurs due to a decrease in the permeability of the intestinal lumen.

As examples of diseases capable of causing chronic surgical constipation, let us cite:
benign and malignant tumors, for example, adenocarcinoma of the lower descending colon, a giant polyp, etc., all processes that produce intrinsic obstruction. Let us cite, on the other hand, as examples of extrinsic compression, some ovarian cysts, uterine fibroids and any extraintestinal tumor, up to physiological pregnancy.

Let us point out as processes capable of determining the symptom of functional constipation of a neuromotor type, generally of a medical nature, paralytic ileus, the so-called chronic habitual or atonic constipation, as well as spastic constipation and rectal dyschesia.

Of a nervous nature, we have constipation due to meningitis, brain tumors, spina bifida neuroles, myelitis and spinal trauma, among others.

To conclude, let us mention the presence of constipation in cases of dry and low-residue diets, in convalescence and in the state after fasting, as well as constipation due to violation of defecatory hygiene, constipation that follows the use of purgatives and that of pregnancy, and that occasionally determined by a psychic alteration (anguish or fear), more frequently psychoses and psychoneurosis depressive type.

DIARRHEA

Concept

The diarrhea symptom is defined as an increase in the number and quantity of normal periodic bowel movements. It consists of the too rapid evacuation of excessively fluid stools (Roux) or the expulsion of abnormal products from the intestinal mucosa.

As can be seen, the qualitative alteration of excrement is the rule in diarrhea, in addition to the increase in the quantity and number of bowel movements that are normally made every 24 hours.

In identifying diarrhea, it is important to know the patient's habit, since some subjects normally move their belly 3-4 times a day without substantially modifying the character of the excrement.

On the contrary, diarrhea may occur even with a single daily bowel movement if there is evident fluidization of the excrement. Let us particularly point out once again, the existence of the so-called false diarrhea" by Mathieu, cited in connection with the study of the constipation symptom. For us, in reality, it is a true diarrhea, since although it sometimes hides constipation due to fecal impact or rectal neoplasia, it is nothing more than a concomitant diarrhea, produced by the alteration of the neighboring rectal mucosa, one of the pathophysiological mechanisms that we will study in this symptom. Of course, in this so-called false diarrhea, hard fragments of ex- Crement with mucus and products of the rectal wall.

As synonymous terms, patients report that they have " belly decomposition", "stomach decomposition, that they are "loose in the belly", etc.

The frequency of this symptom is great and its presence can mean either a particular intestinal digestive disease or a disease of any other apparatus or system of the economy. It is noted that if the histories of patients of all kinds are generally reviewed, the symptom diarrhea in adults could be present as frequently as 50-55%. This includes accidental diarrhea, acute diarrhea, and chronic diarrhea. Chronic diarrhea is reported by Fradkin to have a general frequency of 10%. In Cuba, these figures seem acceptable and those caused by neuromotor and inflammatory alterations are now considered the most frequent diarrhea.

Semiogenesis or pathophysiology

It is understood that we will expose the pathophysiology, both of acute diarrhea and especially of chronic diarrhea, the digestive and extradigestive causes; We will do this simultaneously, analyzing the different semigenetic factors. Let's first list them.

- Alteration of the intestinal mucosa.
- Alteration of neuromotor balance.
- Decrease in enzymatic action.
- Decreased intestinal absorption.
- Dietary alterations.

Alteration of the intestinal mucosa

There are numerous circumstances in which the offending agent alters the intestinal mucosa at a certain level, to such a degree that the products of inflammation of the mucosa are incorporated into the excrement, fluidizing it, accelerating transit and producing what is known as organic, parietal diarrhea, an index of a more or less marked alteration of the intestine itself.

Sometimes this alteration is so marked that the pathological elements of the mucosa: mucus, blood

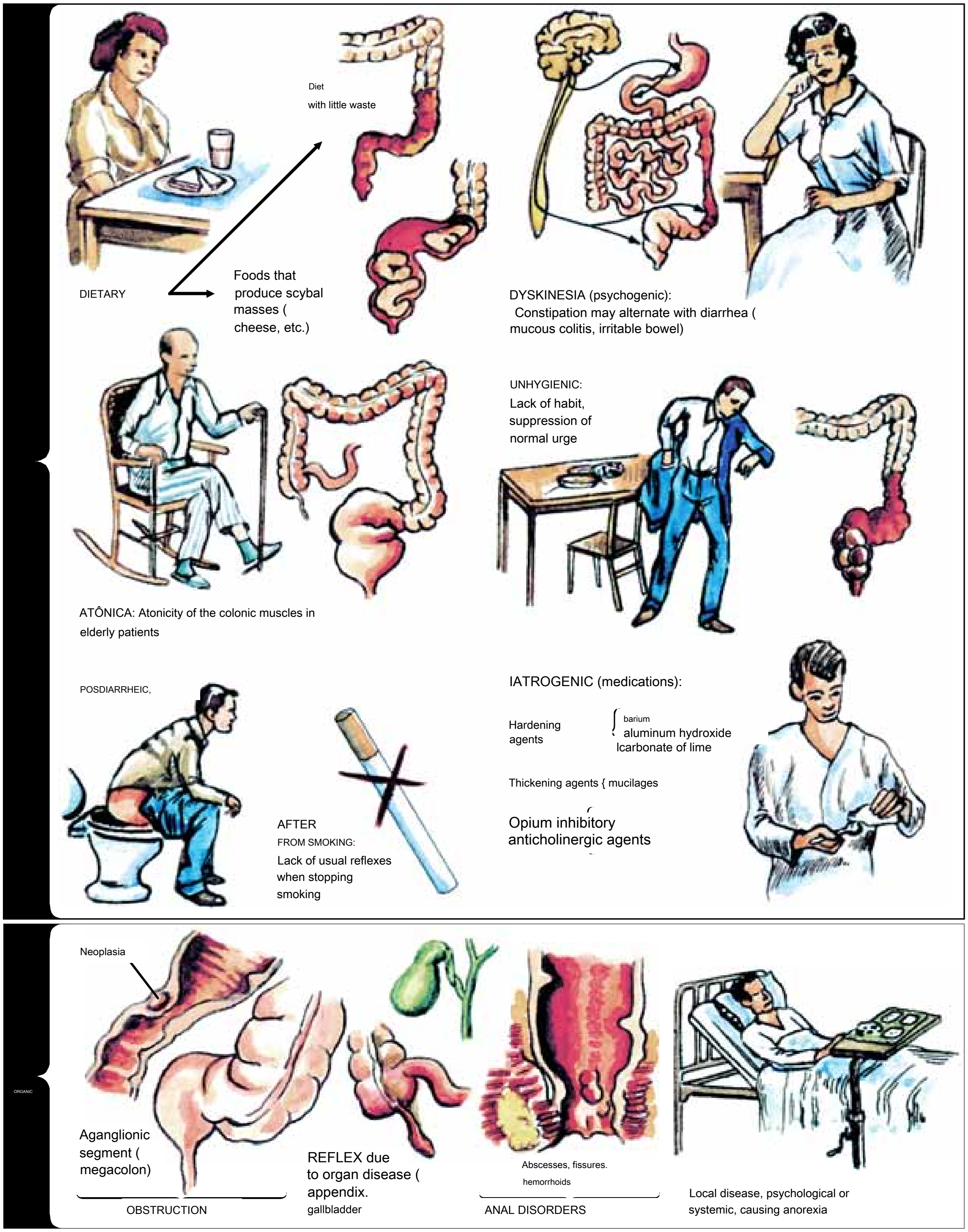

Diet
with little waste
DIETARY
Foods that produce scybal masses (cheese, etc.)
DYSKINESIA (psychogenic):
Constipation may alternate with diarrhea (mucous colitis, irritable bowel)
UNHYGIENIC:
Lack of habit, suppression of normal urge
ATÔNICA: Atonicity of the colonic muscles in elderly patients
POSDIARRHEIC,
IATROGENIC (medications):
Hardening agents
barium
aluminum hydroxide
lcarbonate of lime
Thickening agents { mucilages
Opium inhibitory anticholinergic agents
AFTER
FROM SMOKING:
Lack of usual reflexes when stopping smoking
Neoplasia
ORGANIC
Aganglionic segment (megacolon)
OBSTRUCTION
REFLEX due to organ disease (appendix.
gallbladder
Abscesses, fissures.
hemorrhoids
ANAL DISORDERS
Local disease, psychological or systemic, causing anorexia

Fig. 58.2 Semidiagnosis of constipation.

epithelium, etc., constitute per se, the diarrhea itself, particularly if the portion of the affected mucosa is the distal one, that is, in the terminal portions (rectosigmoid). In turn, let us point out within this heading of irritative diarrhea, two fundamental mechanisms:

1. Inflammatory irritation mechanism, which corresponds to the histopathological response of the mucosa to infectious or parasitic processes. These processes can be: jejunal, ileal and colonic, depending on the location; That is why they are known by the terms jejunitis, ileitis, colitis and enterocolitis.
2. Mechanism of irritation due to neoformations and abnormal infiltrations of the mucosa. Tumors, especially malignant ones, incorporate the products of their disintegration into the stool caused by necrosis and secondary infection, causing diarrhea of this type.

The alteration of the intestinal mucosa causes organic diarrhea. Practically, all the remaining mechanisms that we will study below determine the so-called functional diarrhea.

Neuromotor balance disturbance

We already pointed out the neuromotor mechanism of constipation. In the same way, there is also neuromotor diarrhea, which is due to the alteration of any of the factors that regulate normal motility. Motor diarrhea is due to a modification due to hyperdynamia, motor excitement or hypermotility:

a) If the neural balance is altered and the cholinergic system predominates over the adrenergic system, precisely the opposite of what occurs in the constipation symptom.

b) Neuromotor diarrhea is also observed due to exaggeration of the gastroileal and gastro-colic reflexes, in general, as a consequence of hyperreflexia from certain lesions acting as irritating thorns: vascular lesions of the abdomen:; A tumor lesion of the right colon that is non-obstructive and incapable of producing diarrhea due to parietal alteration, may be the cause of motor diarrhea, exciting peristalsis at that level.

c) Also, for different reasons, peristaltins can increase, or normally inhibitory substances decrease, and hypermotility with motor diarrhea is also established. We can point out the hormonal action, for example, of the thyroid in hyperthyroidism, the action of bile in dyskinesias and biliary fistulas, with the contribution of true bile spills in the upper portions of the intestine.

d) In cases of rapid transit due to pathological short circuit or due to operative acts, rapid emptying of the stomach or duodenum and fistulas, particularly gastrocolic.

e) In constipation itself, when the neurovegetative and intrinsic neural imbalance alternate, going from an adrenergic predominance to a cholinergic predominance that produces diarrhea, as occurs in mixed dyskinesia of the colon, the so-called irritable colon.

f) Sometimes, as occurs with constipation, an alteration of the central nervous system: spinal cord or brain may be the cause of diarrhea.

g) Finally, within this great neuromotor mechanism, we point out with great interest motor diarrhea of a psychological or emotional nature. Grace and Wolf carefully studied this diarrhea in subjects with fistulas due to surgical interventions carried out for curative purposes and confirmed ad nauseam, the hyperdynamic diarrheal reaction with increased propulsive power, hyperemia of the mucosa, tapering and contracture of the circular muscle fibers, which corresponded to periods of mental irritation of the subjects: discomfort, disgust and tendency to hostility and anger. Let us note in passing that the opposite response, hypodynamic, rather corresponds to the phases of depression, anxiety, fear and dissatisfaction. The use of cholinergic agents also caused motor diarrhea in these subjects.

Decreased enzymatic action

The production of diarrhea is evident due to lack of digestion of one or more of the chemical food components of the diet. With the decrease or absence of ferment, diarrhea occurs characterized by the presence of remains of unattached food, which in turn irritate the mucosa and secondarily determine a motor reflex. There is acceleration and easy fluidization of the excrement. There are many examples, but let us mention among them: diarrhea rich in neutral fats of pancreatopathies with marked deficiency of pancreatic lipase, due to lack of breakdown of lipase into fatty acids and glycerin; Another example is diarrhea due to amylolytic deficiency, etc.

Decreased intestinal absorption

Foods can normally be attacked by enzymes, but without absorption of the disintegrated elements. For example, in children there is celiac disease, in which the fatty acids produced by the normal breakdown of neutral fats are not absorbed due to mucosal alterations caused by gluten allergy. Other similar processes are sprue and adult spuriform syndromes. Sometimes, it is an injury to the quills due to: tuberculosis, infiltrations by fat or other pathological substance, which determines diarrhea due to intestinal absorption deficiency. The diarrhea caused by this factor makes up the intestinal malabsorption syndrome, which we will study later.

Feeding disorders

Occasionally or permanently, a diet altered by excess fluids, increase in cellulosic residues or unevenness in the particular proportion of carbohydrates, proteins or fats, can secondary cause irritation of the mucosa, motor alteration or digestive overload, which is an obvious reason for diarrhea.

semiography

We will analyze the following factors, which are listed in table 58.3:

1. Periodicity. In many cases the periodic nature of the diarrhea helps in the diagnosis. Mucomembranous or neurogenic colitis, emotional diarrhea, ulcerative colitis, regional ileitis, diverticulitis and some gastrogenic diarrhea are intermittent over a more or less prolonged period of time, in which cases the patient can go for periods of time without diarrhea. In other cases, diarrhea is constant, permanent from its appearance, as occurs in colon carcinoma and tuberculosis, for example.

2. Hourly rhythm. There are early postprandial diarrhea; that is, they occur immediately after meals due to exaggeration of the gastrocolic reflex. This group includes acute colitis, idiopathic colitis, and segmental enteritis. Gastrogenic diarrhea is also of this type, as is episodic biliary diarrhea, which is observed in some cholecystectomized patients. Emotional diarrhea frequently presents this rhythm or schedule.

Table 58.3

Semiographic characteristics of I to diarrhea
1. Periodicity 2. Rhythm or schedule 3. Intensity 4. Quality 5. Start mode 6. Way to calm down 7. Associated symptoms 8. Course

3. Intensity. The intensity of a diarrheal process is calculated by the number of stools and the quantity.

Profuse diarrhea is generally due to enzymatic or absorption disorders. They are produced at the expense of the large nutritional content that is poorly attacked or not absorbed. They are commonly of high origin; of the stomach itself, the duodenum, the jejunum or the ileum, which is why they are classified in daily clinical language as high diarrhea. In turn, usually the number of bowel movements is not considerable, ranging from 1 to 6 or 8 at most in 24 hours. Sprue diarrhea sometimes presents stools weighing 1,000 g.

Low-quantity diarrhea is due to irritative processes of the mucosa, generally in the lower portions of the intestine. They are dysenteric diarrhea, due to rectosigmoiditis, in which the stool sometimes resembles sputum. Its content is mucus and blood, its number is considerable, 20, 30 and even 50 can be emitted in 24 hours, in certain cases of bacillary dysentery, for example. These are what we call low diarrhea in daily clinical practice. They are also observed in amoebic dysentery, idiopathic ulcerative colitis, etc.

4. Quality. The character of the bowel movement reported by the patient is also important. Apart from the quantity, the color, the smell, the presence of phlegm, blood, parasites and foreign bodies, they can guide the semiotician as an advance in the judgment that will be made in greater detail by the laboratory technician. It is important to know if the patient has ingested substances or foods that can color feces, such as beets, blood sausage, bismuth, charcoal, among others. Clear bubbly diarrhea indicates fermentative processes as in sprue, dark diarrhea indicates putrefaction, and yellow diarrhea indicates the presence of bile.

Rancid smell indicates fermentation; On the contrary, stench indicates putrefaction.

Diarrhea with a mucopousanguineous flow indicates organic damage to the terminal portion, as occurs in the dysenteries, so often cited. The presence of food remains indicates lienteric, gastrogenic diarrhea, for example. The presence of isolated abundant mucus, sometimes enveloping a fairly normal and even hard stool, is typical of mucorrheic colitis and irritable colon, as well as villous tumors. Sometimes the diarrhea is a fairly solid paste, as occurs in steatorrhea (fatty neutral) of pancreatopathies. In short, this is the clinical study of the stool, the urinal or tank in which the patient defecates, and whose study we always recommend in a similar way to the examination of the diaper by the pediatrician, without this meaning that we do not study diarrhea through the laboratory through the comprehensive coprological examination, as a complementary diagnostic resource.

5. Start mode. Any diarrhea of sudden onset that leads to acute inflammatory processes, infectious or toxic diseases or sudden transgressions of the dietary regimen constitutes acute diarrhea. On the contrary, if the onset is slow, one thinks of chronic diarrhea, functional processes of different origin or organic processes of slow establishment.

6. Way to calm down. If the diarrhea symptom disappears spontaneously or with few therapeutic measures, one thinks of accidental diarrhea due to transgression, of emotional diarrhea, but if it resists treatment, becomes rebellious, one should think of a permanent functional cause, such as an enzymatic or absorption insufficiency, or an organic process, such as a tumor, or ulcerative colitis.

7. Associated symptoms. As always, they are a great value. We can observe the associated diarrhea, alternating with constipation. This is typical of irritable bowel syndrome. We have also pointed out the association of constipation in the so-called false Mathieu diarrhea, or inflammatory diarrhea secondary to irritation of the rectal mucosa in obstructions produced by tumors or rectal impact. The presence of fever indicates infectious processes, especially bacillary dysentery, salmonellosis, etc.

The association with pain is of great importance in guiding the semiographic value of diarrhea, since pain means the presence of peristaltic alteration and irritation of the nerve fibers of a certain region of the intestine. Painful diarrhea guides towards the diagnosis of enteritis, in particular, ileitis and more or less acute colitis. Painful diarrhea also guides the diagnosis of tumors, especially malignant ones. If the associated pain has characteristics of tenesmus and straining, this implies the low, possibly rectal location of the diarrheal process.

On the contrary, an absence of pain will suggest functional diarrhea due to enzymatic or absorption alteration, as occurs in the diarrhea of sprue or pancreatic insufficiency. Similarly, episodic biliary diarrhea, as well as emotional diarrhea, are generally painless.

The presence of associated anal burning indicates the fermentative nature of diarrhea. Abdominal distension and gas are the rule in fermentative diarrhea, spuriform diarrhea, or some partial obstruction syndromes with diarrhea. The one associated with a mass or tumor in the belly is obvious in its diagnostic significance. The presence of hives and migraine will point to allergic diarrhea. This diagnosis also guides the relationship between the appearance of diarrhea, allergic manifestations and the ingestion of a certain medication or food. The presence of edema, anemia and glossitis is characteristic of diarrhea due to nutritional deficiency. Finally, the association of diarrhea with the presence of a rich symptomatic procession of palpitations, anxiety, nervousness, etc., will guide towards the psychovegetative nature of diarrhea, without forgetting that it can be a manifestation of hyperthyroidism.

8. Course. Sometimes diarrhea occurs occasionally in a subject, caused by a violation of the regimen, or an emotion. Other times, although diarrhea recurs, it does so without a defined evolution, its course is irregular, capricious, variable, which forces psychovegetative alterations to be considered as the cause. On the contrary, if the course of this symptom corresponds to something defined, with more or less precise clinical characteristics, we consider the existence of an organic or functional cause, but of diarrhea of defined etiology. This is the case with the course of diarrhea due to sprue, chronic ulcerative colitis or regional ileitis, among other examples.

Semiodiagnosis (fig. 58.3)

It is relatively easy, even by deduction, to now list the main diseases capable of producing diarrhea. It is less easy, however, to make the precise diagnosis in a given case.

1. When there are mucosal alterations, inflammatory or from another cause, you should think about:

   a) Bacterial, parasitic, viral, fungal infections, such as bacillary or amoebic dysentery; enteritis due to salmonellosis, colibacillosis, viral conditions, etc. Certain parasitic diseases such as giardiasis, trichocephaliasis, and others. Intestinal mycoses and intestinal tuberculosis should also be mentioned. In Cuba, at the beginning of each summer, we suffer diarrheal outbreaks of relative benignity, classified in popular language as "mayito" or "chiflido", whose infections, in our opinion, do not correspond to a single bacterial pattern, but rather a variable one, according to the focus of the subsoil removed by the rains (B. coli, B. paracoli, salmonella, viruses, etc.).

b) Exogenous and endogenous poisoning such as mercurial and bismuth poisoning in the course of treatments, alcohol, food poisoning, botulism, as well as endogenous poisoning from uremia and decompensated diabetes.

c) Idiopathic ulcerations such as idiopathic ulcerative colitis and regional iletis.

d) We must also point out benign tumors and especially malignant ones, diverticulosis and polyposis.

2. By another functional mechanism, of a neuromotor nature, the following are causes of diarrhea: hyperthyroidism, medullary thyroid carcinoma, irritable colon, emotional shocks, upsets, nervous tension, etc.

3. Diarrhea from pancreatitis, pancreatic cancer, gastritis, duodenitis, sprue, childhood celiac disease and diarrhea from lipophagic granulomatosis should be taken into account when there is a decrease in enzymatic action or absorption.

4. Finally, by dietary alteration we mention diarrhea from accidental indigestion, violations of the diet, etc.

SMALL SYMPTOMS 0 DYSKINETIC SYMPTOMS

The small symptoms of the enterocollicorrectal segment

Are:

- Abdominal distension or bloating.
- Borborygmos.
- Expulsion of gases through the anus.
- Colic heartburn.

ABDOMINAL DISTENSION OR METEORISM

Concept

It is defined as the sensation of fullness and abdominal tension that patients experience, more or less diffusely, generally caused by gases accumulated in the intestine. It is identified in patients because, sometimes, they loosen the belt to relieve the discomfort. Sometimes an erroneous synonym is used when patients express that they have inflammation", an anatomopathological term with different meanings. They also speak of "gas filling".

Semiogenesis or pathophysiology

The intimate, direct mechanism is the accumulation of gases in the intestine with distention of the abdominal cavity. In turn, this accumulation of gases can be due to

wax:

1. Obstacle in the intestinal lumen, in which case the gases accumulate behind the partially or totally obstructed site, following Von Wahl's law, by which when an obstruction occurs at a certain level of a hollow organ, the portions preceding said obstruction immediately dilate. Depending on the level of the obstruction and its degrees, a partial or total distension will be established with subsequent abdominal distention. Obstruction can be caused by multiple causes.

2. Excess gas production due to fermentation or putrefaction, which causes them to accumulate with more or less intensity, causing abdominal distension. This phenomenon may coincide with the expulsion of gas through the anus, expressed by another symptom known as flatulence.

3. Excess gas corresponding to swallowed atmospheric air, in processes of intense aerophagia with aerogastria and aerocolia, generally associated with multiple psychovegetative
4. Absorption deficiency for gases, with their accumulation and consequent abdominal distention. This may be due to diseases that inhibit or destroy the elements that govern normal absorption. In these cases a vicious circle is established, since the greater the intestinal distention, the lower the absorption capacity of the intestine.

5. Electrolyte imbalance. Humoral electrolyte balance plays a large role in intestinal distension. Potassium stimulates the muscle fiber of the intestine and in cases of marked hypokalemia there is great distension, which constitutes paralytic ileus.

Semidiagnosis

We can easily deduce, as a consequence of the analysis of each mechanism, the diseases in which the symptom of abdominal distension may exist:

1. Intestinal obstructions due to benign and malignant tumors, volvulus, obstructions due to flanges or adhesions, etc., all of which we point out for the study of the so-called surgical constipation.
2. Intestinal paralysis due to peritonitis and potassium or vitamin deficiency.

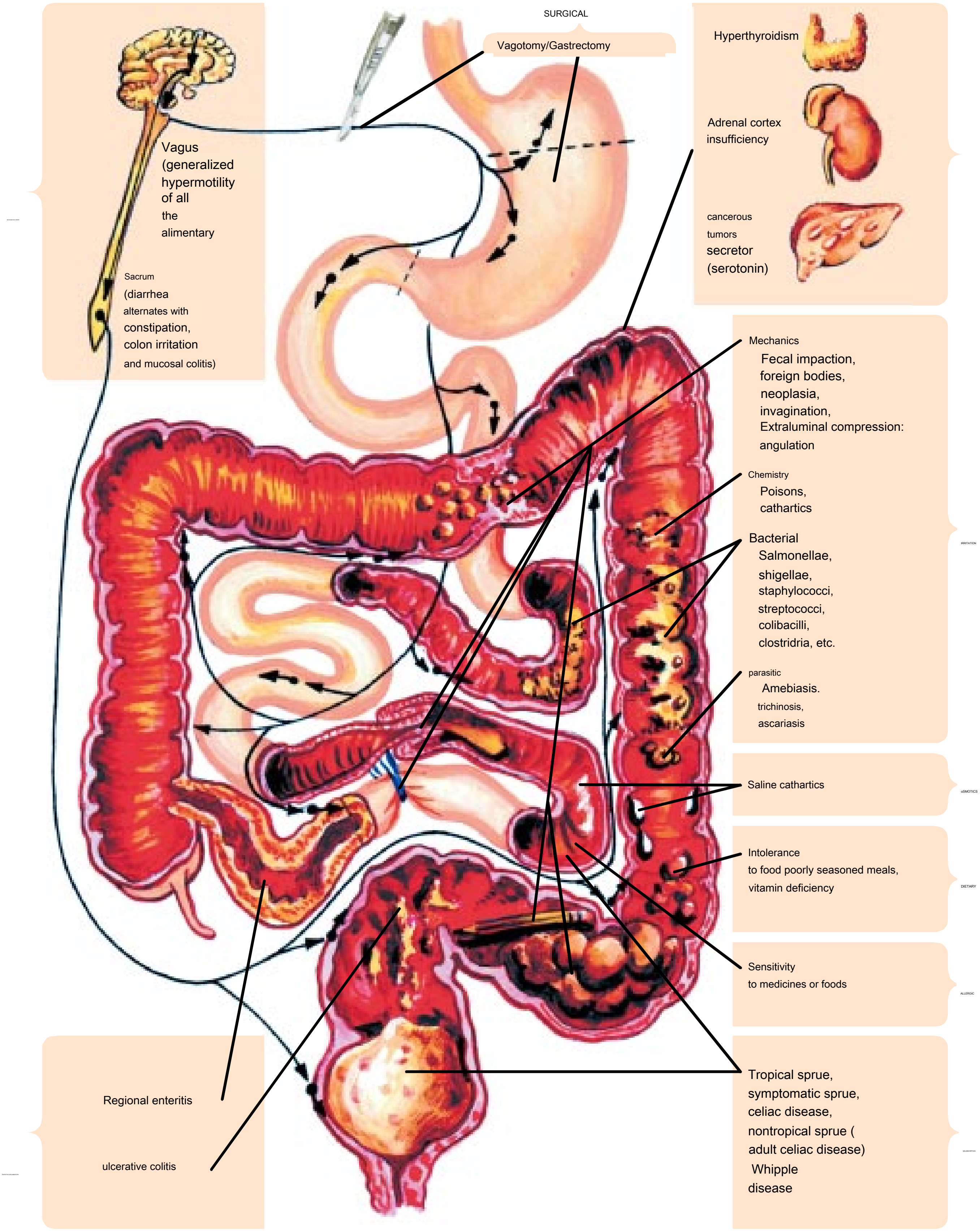

SURGICAL
Vagotomy/Gastrectomy
NEUROMOTOR ORDER
Vagus (generalized hypermotility of all the alimentary
Sacrum (diarrhea alternates with constipation, colon irritation and mucosal colitis)
Hyperthyroidism
Adrenal cortex insufficiency
cancerous tumors secretor (serotonin)
Mechanics
Fecal impaction, foreign bodies, neoplasia, invagination, Extraluminal compression: angulation
Chemistry
Poisons, cathartics
Bacterial
Salmonellae, shigellae, staphylococci, streptococci, colibacilli, clostridria, etc.
parasitic
Amebiasis. trichinosis, ascariasis
IRRITATION
Saline cathartics
oSMOTICS
Intolerance to food poorly seasoned meals, vitamin deficiency
DIETARY
Sensitivity to medicines or foods
ALLERGIC
Tropical sprue, symptomatic sprue, celiac disease, nontropical sprue (adult celiac disease) Whipple disease
MALABSORPTION
Regional enteritis
ulcerative colitis
IDIOPATHIC INFLAMMATORY

Fig. 58.3 Semidiagnosis of diarrhea.

3. Intestinal dyspepsia due to enzyme deficiency or alterations in the bacterial flora, such as fermentative dyspepsia and malabsorption syndromes.
4. Dyskinesias with vagotonia and especially with aerophagia followed by aerocolia, which is observed in patients with profound psychovegetative disorders. It is worth mentioning here the so-called "psychic pregnancy", which occurs in many pythiatic women, heavy air swallowers, etc., particularly when the climacteric begins with menopause, favoring the false criterion of pregnancy, which is nothing more than dis- abdominal tension with intense aerocolia.
5. Another group of diseases capable of producing this symptom are sprue, childhood celiac disease, parasitism, especially giardiasis, and cirrhosis, in which abdominal distension precedes ascites (this phenomenon is expressed in the clinical phrase of the French school that the winds precede the rains).

Sometimes, abdominal distension produces distant symptoms, due to a neighboring compressive mechanism, such as cardiac, pulmonary and reflex neurovegetative symptoms.

BORBORYGMOS

Concept

It is defined as the subjective sensation of audible noises by the patient (and sometimes by the doctor) coming from the belly. They are identified as hydro-aerial sounds located in different portions of the abdomen and at the same time mobile within said cavity. Belly noise", "gut noise", etc. are used as synonymous terms.

Semiogenesis or physiopathology

For borborygmi to occur, the collision or collision of gases with liquid inside the intestine and an increase in intestinal motility are essential. As can be seen, abdominal distension must exist as a prior or associated condition. Therefore, most of the causes indicated in distension will be applied in the pathophysiology of borborygmi, with the requirement of the presence of fluid and hyperperistalsis.

This symptom is typically reproduced when a saline-type purgative is administered to a healthy subject, which causes excess gases and fluid, with hyperperistalsis, and as a consequence, hydro-air collision. In turn, depending on the caliber of the intestinal portion where the phenomenon occurs, a hydro-aerial noise of different intensity and tone will be appreciated.

Semidiagnosis

We can refer here to the diseases listed in abdominal distension. Let us also point out, in particular, Koenig's syndrome, characterized by an intestinal stenosis due to a tumor, in which case borborygmi located in the small intestine are common.

EXPULSION OF GASES THROUGH THE ANUS

The suppression of gases normally expelled through the anus is a valuable sign in all obstructive processes, as well as in paralytic ileus. On the contrary, fermentative intestinal dyspepsia causes an excess of fetid gases. It may also be due to the expulsion through the anus of part of the air swallowed by the aerophages. In this case, the stench of the gases is little or none, unlike when intestinal fermentations or putrefactions take place.

COLIC FIRROSIS

It is the particular painful sensation, referred to in the form of burning or burning, predominantly colonic, partial or generalized. It is due to the arrival of intestinal contents to the segment with chronic inflammation of the mucosa, which is why it is almost always not related to the pandrial, they are late and at a fixed time; They appear 6-8 hours after meals, in the case of angular colitis, and at 4-5 hours, in typhlitis.

PARTICULAR SYMPTOMS OF THE RECTUM AND ANUS

The particular study of the pathology of the rectum and anus has developed so much that proctology already constitutes a particular specialty or ultra-specialty within Gastroenterology and Surgery. For this reason it is obvious that we treat separately the group of subjective symptoms of this segment of the digestive system. Without falling into repetitions, we emphasize the details of this semiology.

The most important subjective anorectal symptoms are:

Rectal pain or proctalgia.

- Rectal bleeding.

Rectal flow.

- Anal itching.

Heaviness and rectal protrusion.

-Anal burning.

RECTAL PAIN OR PROCTALGIA

Concept

It is defined as the subjective painful sensation, already described due to abdominal pain, in this case referred by the patient to the anal, perianal, perineal and coccygeal region. We indicate the location with a certain extent, to facilitate overall identification, subject to later knowing its different causes. It is known as synonymous terms "pain in the intestine" as the common people say and as proctalgia, a medical term.

Semiogenesis or pathophysiology

Everything explained in connection with the study of epigastric pain can be applied here, although it is important to point out that the anus itself has nervous connections that belong to the nervous system of relationship life. This explains why anal injuries cause great subjective manifestations and psychological repercussions when compared to rectal injuries. In the rectal area, the Mackenzie viscerosensory reflex with its modalities will persist as a mechanism, while in the anal area a somatic neural reflex similar to that observed in brachial neuralgia, for example, will be established.

Semiography

The most important factors are:

1. Location. Rectal pain can be relatively high and deep, as occurs in a rectal tumor in inflammatory rectitis, or be lower in location, as in an anal fissure. Let us particularly mention the pain located in the coccyx or coccygodynia due to a spasm of the paracoccygeus muscles with or without concomitant bursitis. It can irradiate to the hypogastrium and perineum, but irradiation to the sacrum and coccyx is also observed, depending on the location of the lesion.

2. Intensity. It is variable, but according to the anatomophysiological notes on the innervation that we have made, an anal fissure, for example, is much more painful than a tumor of the rectal ampulla, which is sometimes completely silent. Proctalgia fugax, which we will point out in greater detail, is a type of intense rectal pain. A hemorrhoidal thrombosis and a perianal abscess are pains of marked intensity.
3. Quality. She is a guide in diagnosis. Grabbing pain, of heaviness, is seen in the tumors close to the anus. It has a special character in acute inflammatory processes and is expressed as tenesmus and straining.

   Sometimes, rectal pain appears in a healthy subject, as something surprising, in the middle of sleep, at night, as something intense, deep, above the anal level, as if the patient's rectum was distended with a balloon, the pain reaches paroxysm in minutes and subsides spontaneously or when compression is applied to the buttocks. This corresponds to the so-called proctalgia fugax, which is supposed to be caused by an idiopathic rectal or rectosigmoid spasm; It occurs in subjects with instability of their psychovegetative system, with great nervous tension.

   Rectal pain is generally exacerbated by defecation or only occurs when defecating, which indicates that the cause is located close to the anus: hemorrhoids, fissure, for example.
4. Associated symptoms. The semiological assessment of rectal pain depends a lot, as is logical, on the associated symptoms: constipation, diarrhea or other intestinal symptoms.

Semidiagnosis

Let us particularly mention the pain located in the coccyx, coccygodynia, due to a spasm of the paracoccygeus muscles, with or without bursitis.

Any reflexogenic lesion located in the rectum and anus will determine this pain; Therefore, practically all rectal pathology must be considered. Let us point out, however, the most common diseases:
rectitis of different nature; hemorrhoids in all its degrees; tumors of the rectum and anus, benign and malignant; fissures, ulcerations, fistulas, abscesses, cryptitis, papillitis and rectal endometriosis.

RECTORRHAGIA

Concept

It is nothing more than a modality of enterorrhagia with a frankly rectoanal location. It is defined as the expulsion of red, fine, sparkling blood from the anus, which appears to come from a short distance. Many times the patient identifies this hemorrhage when it is discreet, by staining the toilet paper with which he wipes himself after defecating red, and if it is more intense, by seeing fresh blood in the toilet bowl.

Semiogenesis or pathophysiology

We will not fall into repetitions with the exposition of semigenetic knowledge that is common to enterorrhagia in general. We only point out, redundantly, that the essential pathophysiological condition is that the hemorrhagic lesion is located in the rectum or anus.

Semidiagnosis

The diseases that most frequently cause rectal bleeding are: hemorrhoids and rectal carcinoma, whose differential diagnosis is very necessary due to the importance and frequency of both diseases. Usually a simple rectal examination by experienced hands clarifies the diagnosis in a timely manner. Polyps also bleed, as well as hemorrhagic rectitis of a different nature, particularly ulcerative colitis of this location. Let us also point out rectal endometriosis, a disease that we must always take into account when faced with rectal bleeding in a woman with associated menstrual disorders.

RECTAL FLOW

Concept

It is defined as the expulsion through the anus of mucopurulent and purulent mucous secretions. Rectal discharge is a symptom that easily expresses the manifest inflammatory alteration of the terminal mucosa. The patient identifies his discharge by running down his thighs or staining his clothes. The secretion varies in quality, color and appearance depending on its nature. It can also come out mixed with blood. It is important to check if the flow comes out through the anus or through some small hole that the patient suspects of having on the margins of the anus, which would correspond to open fistulas.

Semiogenesis and semidiagnosis

Rectal flow is observed in septic rectitis, particularly in rectal lymphogranulomatosis or Nicolás and Favre disease, in those of gonococcal origin (nowadays much less frequent), in abscesses and phlegmonous processes of the rectum and anus, as well as in fistulas subsequent to these last two lesions or of congenital origin.

ANAL ITCHING

Concept

It is defined as the sometimes unbearable sensation of itching in the anus and surrounding region. This symptom has great proctological importance, since sometimes the intensity, intolerance, psychological repercussion and difficult cure pose situations of great responsibility to the proctologist.

Semiogenesis or pathophysiology

In this symptom, an irritability of the rich nervous system of the region is established, with reflexology constant to the nervous centers, which can occur due to multiple causes or injuries that act as an irritating thorn. We must first accept the secondary type itch that we have pointed out, but in many cases, the itch is primitive or essential and develops on a psychosomatic background.

Semidiagnosis

In its semiological significance, let us point out as pruritic rectoanal diseases: oxyuriasis, in which cases the female's egg laying on the margins of the anus determines the itching; hemorrhoids; small erosions or fissures; papillitis and cryptitis, eczema, among others. Pruritus is generally established by nocturnal exacerbation attacks. Let us also point out the anal itching of diabetes and allergy, to talk again about the idiopathic, neurogenic or psychosomatic anal itching, where the individual factor is fundamental, determined by itching and subsequently aggravated by anatomical changes in the region, sometimes irreversible. Dermatological diseases of the region (lichen planus, eczema, etc.) should not be forgotten.

RECTAL HEAVYNESS AND PROTRUSTION

Concept, semiogenesis and semiography

It is defined as the sensation of something heavy or coming out of the anus. The patient goes to the doctor to have the region examined and he generally finds tumors, hemorrhoidal masses, rectal prolapse, condylomas, granulomas, etc.

ANAL BURNING

Concept, semiogenesis and semiography

It is defined as the burning sensation, sometimes even a burning sensation, that patients experience on the margins of the anus.

Its semiogenesis corresponds to the contact of an irritating fecal material with the anorectal mucosa, which in turn may have been previously irritated by inflammation or erosion.

The semiological significance of anorectal burning indicates as the main diseases fermentative diarrhea, any excrement of acid reaction, proctitis of different origin, as well as any erosive, inflammatory or other lesion of the terminal intestine. It is also caused by diarrhea with a large bile content.

DIGESTIVE SEMIOLOGY IN PHYSICAL EXAMS

GENERAL AND REGIONAL

59

DIGESTIVE SEMIOLOGY IN THE GENERAL PHYSICAL EXAMINATION

Following the anamnesis or interrogation, a general physical examination of the patient should be performed. The exploration techniques that we use for this are the same ones that we studied in volume 1, Section I, Chapter 3.

The most important semiological aspects of the digestive system that can be detected in the general physical examination are found in the exploration of:

Attitude and recumbency.
- Constitution.
- Weight and size.
- Facies.
- Skin color.
- Extremities.

ATTITUDE AND DECUBIT

In some patients, the attitude they adopt becomes a physical sign of courage. Dorsal recumbency is observed in most acute inflammatory painful processes of the abdomen, especially in appendicitis and peritonitis. The patients remain motionless face up, in bed, with their lower limbs semiflexed.

Prone ventral decubitus is observed in processes characterized by intense, diffuse abdominal cramps, such as those corresponding to acute colitis, tabes and especially lead colic; In this disease, the patient causes great compression of the belly with his fists or any object. The case, described by Stochusen, of the painter from Angers is cited, who relieved his saturnine colic by inviting two or three men to lie on his belly. Some cases of anterior piloroduodenal ulcer also adopt the prone position.

The standing attitude is cited, with the body bent forward (Cheinisse). in some cases of posteriorly located gastroduodenal ulcer accompanied by perivisceritis. In neoplasia of the body of the pancreas, this attitude is very common, as is the sitting position leaning forward or the prone position, attitudes with which they achieve relief and which have great semiological value (fig. 59.1).

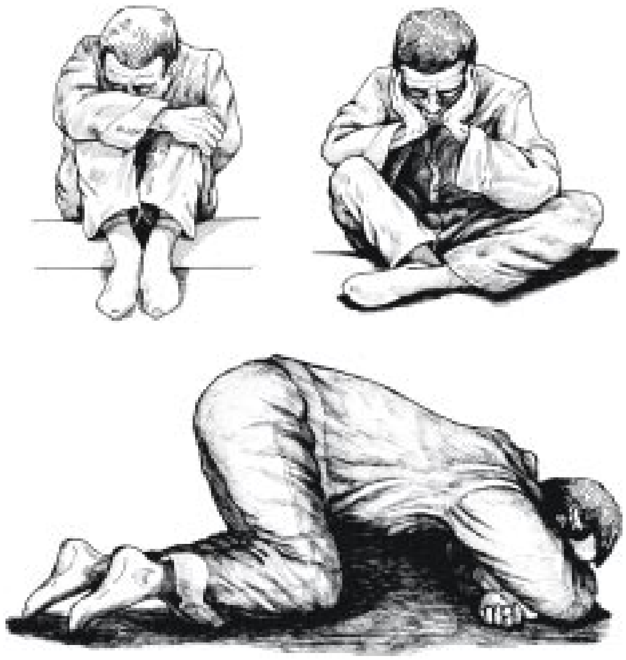


Fig. 59.1 Patient's attitude in pancreatic neoplasia.

In perivisceritis, as well as in acute cases of hernia strangulation, patients adopt special, highly variable and capricious attitudes in bed (lateral decubitus and others), with the idea of escaping the compression or stretching of adhesions, flanges, etc., which cause pain.

Finally, sometimes the patient's gait is altered in digestive processes. In cirrhosis with extensive ascites, the patient's gait is typical, reminiscent of that of a full-term pregnant woman (compensation lordosis).

CONSTITUTION

We already know from the studies of General Semiology, in volume 1, the different constitutional types of the Italian, French and German schools. The identification of the constitutional type, through the general physical examination, has application in digestive semiology to guide the diagnosis.

The longilinear and asthenic type (leptosomal, microsplanchnic, etc.) rather suggests a functional digestive pathology, linked to visceral ptosis and psychoneurovegetative imbalances corresponding to gastrointestinal dyskinesias and many stomach ulcers.

The brevilinear type (pycnic, macrosplanchnic, etc.) is more oriented toward organic diseases and diseases of the upper abdomen. Let's mention cholecystopathies, pancreatitis, gastritis, among others.

The existence of specific habits for certain diseases is evident; for example, the habit of liver cirrhosis. This habit is manifested by alterations visible morphologies that make up the so-called Chvostek cirrhotic habit, frequently observed in these patients, which suggests that there is a certain predisposition in them for the development of said disease.

Among the most obvious stigmata of the cirrhotic habit we will mention: the poor hair development in the beard, armpits, abdomen and pubis, which sometimes adopts a feminine disposition; umbilical ptosis, which is manifested by a xiphoid-navel distance much greater than navel-pubis, present in 75% of cases; genital hypoplasia, with greater breast development at times and predominance of the length of the lower extremities.

WEIGHT AND SIZE

They are very closely linked to the nutrition of the patient. The decrease in weight indicates global malnutrition, which is why we think of digestive diseases, generally organic, accompanied by malnutrition, such as: cancer, severe colitis (ulcerative colitis), cirrhosis, etc. In other diseases, almost always functional, weight is maintained or increased, as occurs in neurovegetative dyskinesias. Height is altered in cases of developmental arrest due to serious digestive pathologies and its alteration also suggests the relationship of said digestive pathology with endocrine alterations.

In the general physical examination of many patients with the digestive system, we can identify a series of signs that belong to a qualitative nutritional deficiency, which are studied in the alterations of the examination of the mouth, skin, hair, eyes, tibial edema, etc., and which correspond to the different deficiency syndromes of vitamins of the B complex, proteins and others.

FACIES

The peritoneal or hippocratic and hepatic facies have already been studied in this work, in volume 1, Chapter 24.

Hippocratic facies is observed in patients with acute and subacute peritonitis, in severe and complicated colitics (bacillary dysentery, idiopathic ulcerative colitis, etc.), in whose processes dehydration and intoxication are predominant phenomena.

In cirrhosis, the facies is characterized by the projection of the facial mass, paleness, a sharp nose with vascular striae on the lateral parts, especially in alcoholics (Burni and Shuth sign) . In the neck and upper part of the chest, the spider veins so typical in chronic active hepatitis and cirrhosis are observed. In biliary cirrhosis, the icteric greenish color and lipoid skin deposits (xanthomas) stand out, almost always located on the eyelids in the form of round or oval plaques or infiltrates (Fig. 59.2).

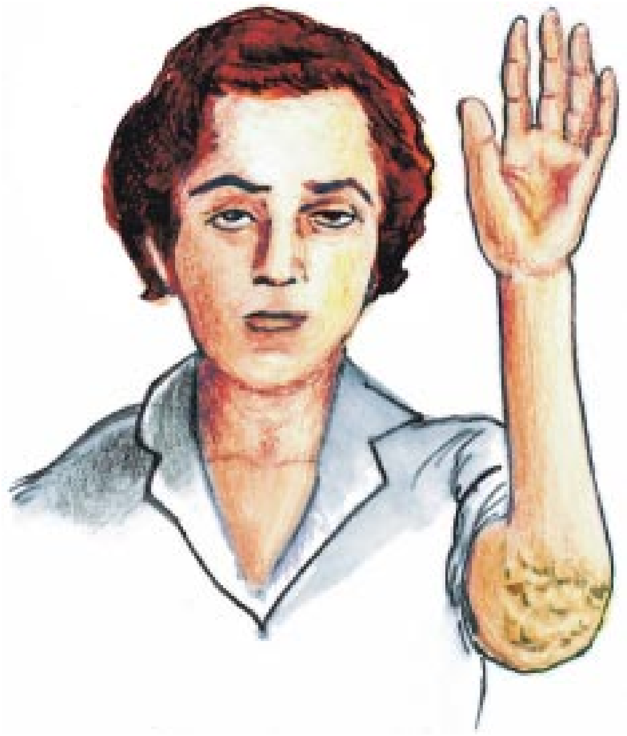


Fig. 59.2 Skin facies and xanthomas in biliary cirrhosis.

It is necessary to point out the facies of the secreting carcinoid syndrome, which appears through crises that can last from a few minutes to half an hour and during which a purple-red, sometimes violet, congestion (flush) occurs on the face and affected body regions. These crises due to the release of serotonin (5-hydroxytryptamine) by the argentophilic cells of carcinoid tumors that almost always occur in the digestive tract, can be triggered by the ingestion of cheese, wine, or by palpatory maneuvers of the tumor.

Although this entity is rare, we point it out due to its typicality, which allows diagnostic guidance only with observation.

SKIN COLOR

This aspect has already been discussed in Chapters 3 and 25, volume 1. The most significant color in the digestive system is the yellow color of jaundice, which will also be studied in Chapter 63 (the syndromes of this system).

LIMBS

The examination of the extremities also offers data of semiological interest.

In the upper limbs, spider veins are common, which we have already studied on the face, true pulsating angiomas with a crown of radiating vessels.

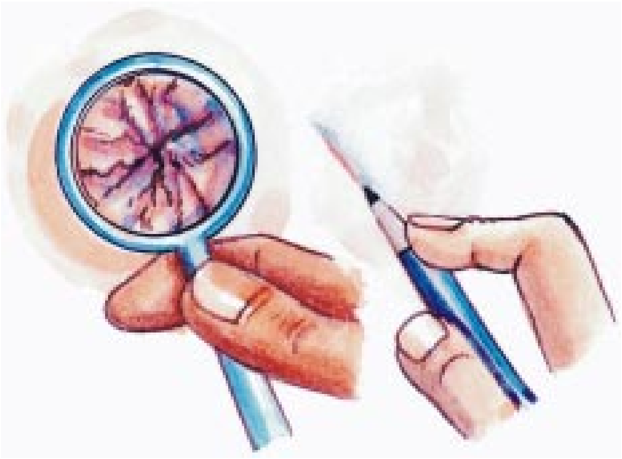


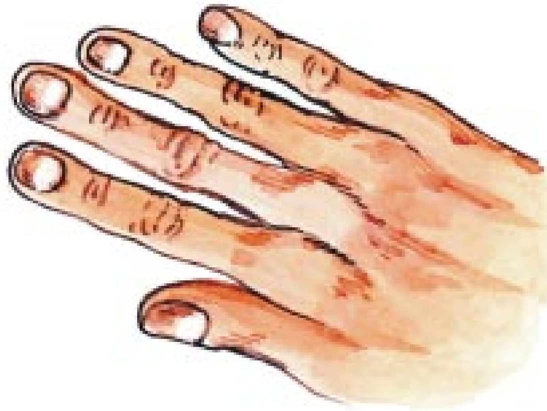
Fig. 59.3 Technique to differentiate spider vein from telangiectasia.

two eccentrics that give it the appearance of a star or spider (a characteristic to which they owe their name: stellar dilations or vascular spiders); They occur due to insufficient neutralization of estrogenic substances in the liver.

Angiomas can be easily differentiated from similar lesions (red moles, telangiectasias) by compressing the central button with the tip of a pencil (or something similar) , with which the peripheral veins disappear, only to reappear when said angiomatous button is decompressed (Fig. 59.3).

On the hands we can find the deformities of the fingers, already pointed out in other parts of this book, clubbing fingers, and the characteristic nails that accompany this sign (fig. 59.4).

Fig. 59.4 Clubbing of the toes and nail bed changes in cirrhosis.



However, alterations of the nails alone can also be found, which have been called nails without lunula, white nails, Terry nails (see Chapter 25, volume 1), which are due to the existing dysproteinemia.

The palm of the hand shows diffuse redness, with redder spots inside, which is preferably located in the thenar and hypothenar regions and on the tips of the fingers. This very characteristic alteration, which has been called palmar erythema or hepatic palms, is constituted by the confluence of multiple angiomas that give it that characteristic pink or violet tint (fig. 59.5).

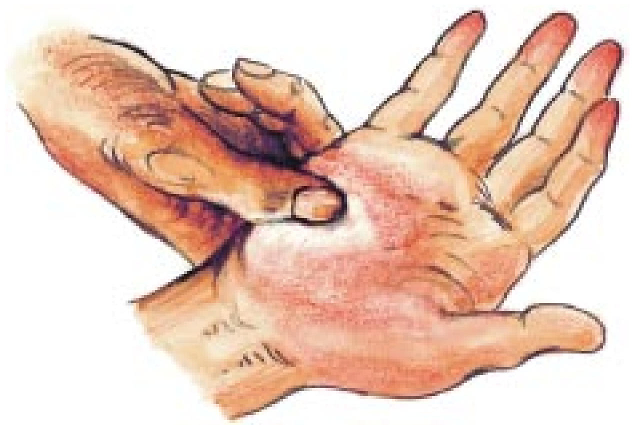


Fig. 59.5 Palmar erythema.

Let us point out that palmar erythema, like the previous alterations, can appear, although much less frequently, in non-hepatic diseases and is even observed in chronic smokers, chronic lung diseases and certain hyperstrinisms.

DIGESTIVE SEMIOLOGY IN THE REGIONAL PHYSICAL EXAMINATION

We must analyze the alterations in the regions of the head, neck, thorax, abdomen and extremities. The digestive semiology in the physical examination of the abdomen will be studied in Chapter 60 and the detectable alterations in the examination of the extremities were already discussed previously.

We will address the alterations of the physical examination, after an anatomical summary and details of the natural physical examination (if they were not done previously in volume 1, Section I, Chapter 16), with the analysis, in as much detail as possible, of the semiography or particular study of each objective symptom and its semiological assessment, to make, when useful, an incursion into the semiogenesis of said symptoms. We will follow a rigorous order in our analytical excursion of the different regions. At the same time, in order not to lose the methodology of this medical discipline, we will go from the symptom to the disease analyzing each one in its different characters: volume, color, loss of substance, shape, and we will never begin with the naming of nosological entities. Of course, we do not intend to point out all the physical symptoms, which would be endless, but rather the most frequent examples in daily clinical practice.

In the head, neck and thorax, we will study changes in the physical examination of the mouth, pharynx and thoracic esophagus.

PHYSICAL EXAMINATION OF THE MOUTH

Introduction

This is an important issue, both for the general practitioner and for the gastroenterologist and the stomatologist, since the multiple alterations of the mouth can correspond to local as well as systemic diseases.

In this aspect of semiology, we have an example of the equivalence of the symptom and the disease, since in many cases by identifying a certain objective symptom, a defined nosological diagnosis can be directly made.

Semiogenesis or pathophysiology

The objective symptoms of oral conditions have an anatomopathological substrate in 90% of cases: however, the pathophysiological substrate is very small. In the etiology and pathological anatomy of oral symptoms, the factors of the general etiology, as well as all general anatomopathological lesions, will always be taken into account. Any acute infection (eruptive diseases, for example), chronic infection (TB, lues), acute and chronic poisoning (Hg, Bi, etc.), allergy, trauma, congenital conditions, etc., can easily be expressed in the mouth and be responsible for the objective symptoms that we will describe later. We will observe: congestion lesions, exudation, suppuration, necrosis, edema, neoformation, ulceration, scarring, etc., as the anatomopathological substrate of this group of symptoms.

One of the examples of the most typical systemic condition in its oral expression is measles, but we can also cite examples of local conditions of the digestive system, such as the coated tongue of a dyspeptic.

Semiography and semidiagnosis

Closed mouth (external examination): lips and corners

We observe pathological volume variations in the lips, generally due to increase. Can

find some common tumors in the region, such as lymphangioma and epithelioma (most common of the lower lip) with exulceration; thick lips due to endocrine disruption, as in acromegalic and hypothyroid patients;
inflammatory type lips, such as upper lip furuncle, insect bite edema, angioneurotic edema, alimentary urticarial edema, among

others.

As an example of lip deformations, let us point out those of the congenital type, such as lip inversion and cleft lip, in the latter the anomaly may be limited to the soft tissues or extend to the hard palate (unilateral or double). Scar deformities due to wounds or burns are also observable.

Another lip alteration is color changes, as occurs in anemia, erythremia, cyanosis (circulatory or respiratory disorders), pigment spots, etc.

Eruptions such as viral cold sores are observed, which is characterized by the presence of a group of transparent vesicles of 1-2 mm, located in the transition zone of the skin and the labial mucosa; streptococcal pyodermitis, secondary roseolic syphilis with its special distribution of circinate papules (Brock's elegant syphilis) and the fuliginosities of febrile processes.

Let us also point out the lip ulcerations corresponding to the syphilitic chancre with its indurated base and its characteristic yellowish color, and the ulcerated epithelioma, as the main ones.

Lip concisions, when altered, present interesting objective symptoms. Let us mention facial paralysis with the marked downward deviation of one of the commissures; mouth ulcers (perlèche) or commissural cheilitis, either due to viral or bacterial infection, or due to hyporiboflavinosis (called cheilosis) (fig. 59.6); the rágade, characterized by multiple relatively deep fissures in the commissures, attributed to syphilis when it adopts a radiated form (Fournier stigmata). Finally, alterations of the lip commissures due to labioglossopharyngeal paralysis.

Jugal mucosa

It is the seat of multiple objective symptoms; Let us remember that both the buccal and buccal mucosa, the pharyngeal and the esophageal mucosa are covered by a stratified squamous epithelium similar to the skin, which gives them resistance to changes in temperature and to food or foreign bodies of greater or lesser duration.

pray.

Color alterations equivalent to those reported in the lips are observed. Among the inflammatory alterations, eruptive or not, are stomatitis in all its degrees: catarrhal, exudative, hemorrhagic, necrotic, etc. and of diverse etiology: bacterial, viral, fungal, and others. Aphthous or maculofibrinous stomatitis is quite common, in which tall or small very painful exulcerations can be observed, of variable number, size and location, yellowish white in color, surrounded by an erythematous halo; They persist for 1-2 weeks and have a tendency to recur. The etiology of aphthous stomatitis is multiple and is related to allergic, viral, bacterial, intestinal, endocrine, etc. factors (fig. 59.7).

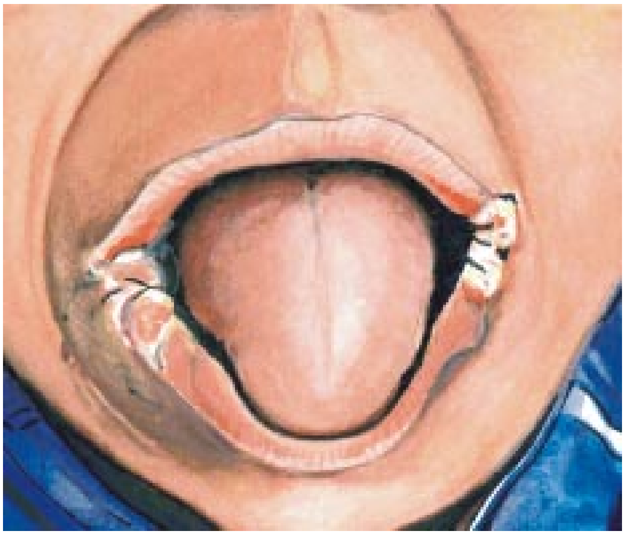
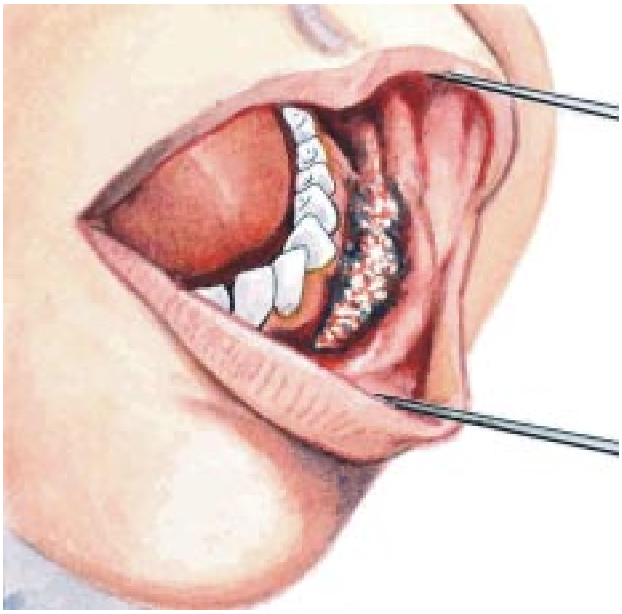


Fig. 59.6 Fissured angular cheilosis.

In eruptive diseases we have: Koplik spots (fig. 59.8) of measles, which are located in

Fig. 59.7 Aphthous stomatitis.



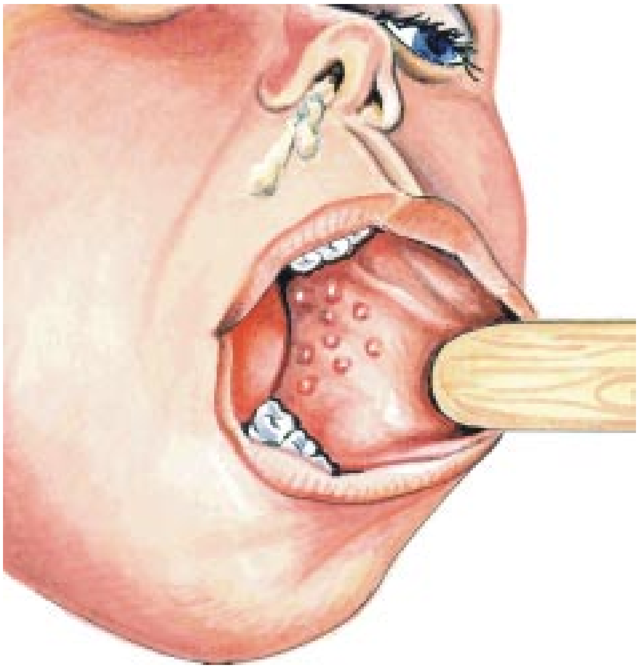
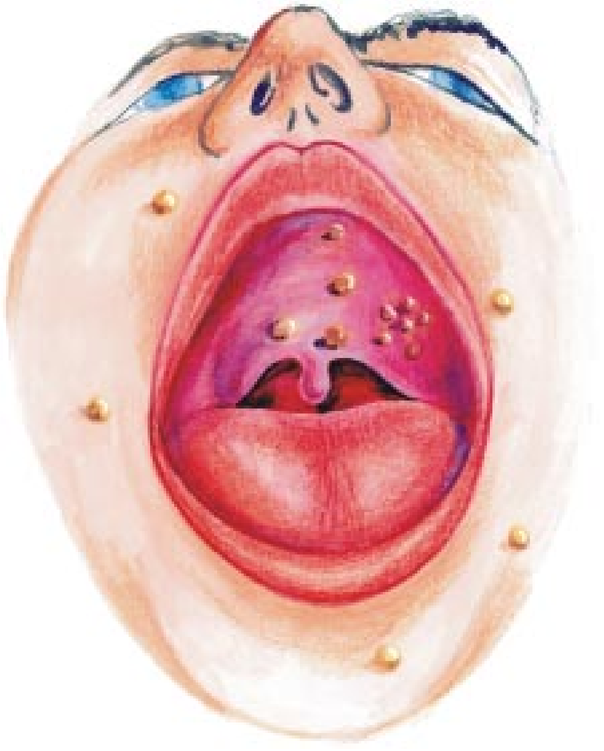


Fig. 59.8 Koplik spots (measles).

The cheeks are whitish in color, with an erythematous halo, appear in the invasion phase, and sometimes constitute a sign of early diagnosis, since it precedes the eruption. The papulovesicular lesions of chickenpox (Fig. 59.9), smallpox, etc.

Fig. 59.9 Papulovesicular lesions (chickenpox).



Other diseases that also affect the buccal mucosa are: thrush (Oidium albicans), common in children and impoverished patients; toxic stomatitis (bismuth, mercury, etc.) (fig. 59.10) and those observed in uremia, diabetes, leukemias and other hemopathies. Among infectious stomatitis, we highlight streptococcal, Vincent's and viral stomatitis. Also considered important are those corresponding to the nutritional deficit (complex B) produced by the local or general use of antibiotics in predisposed subjects.

In chronic adrenal insufficiency (Addison's disease) and in hereditary intestinal polyposis (Peutz-Jeghers syndrome), dark brown or black spots are observed scattered throughout the mucosa that covers the lips, cheeks and gums (Fig. 59.11).

This relationship would be endless, within the limits of this book; On the other hand, it is widely discussed in oral pathology texts.

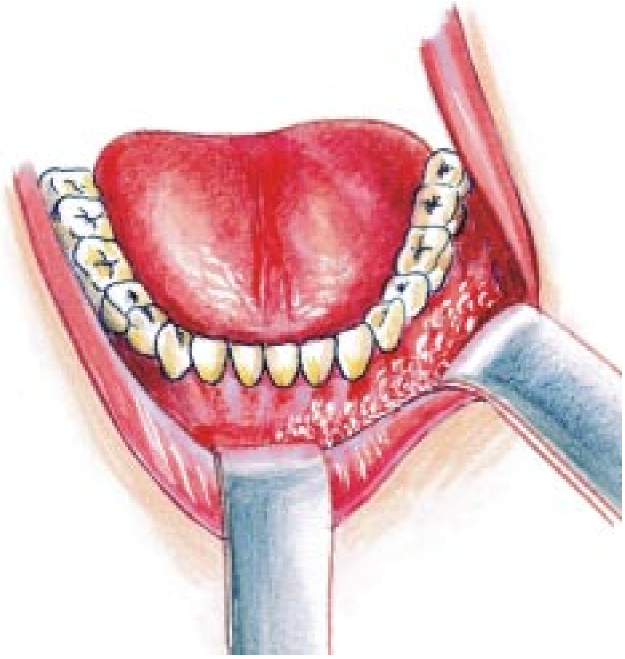
Language

In general, the tongue can express a local alteration, a digestive disorder, or a systemic disease.

The normal language was studied and is illustrated in Chapter 16, volume 1. Here we will study some of the most important semiological alterations of the different types of language.

Volume alterations. The size of the tongue may be smaller than normal, called nicroglossia, which may be congenital or acquired; this alteration is

Fig. 59.10 Toxic stomatitis (mercurialism).



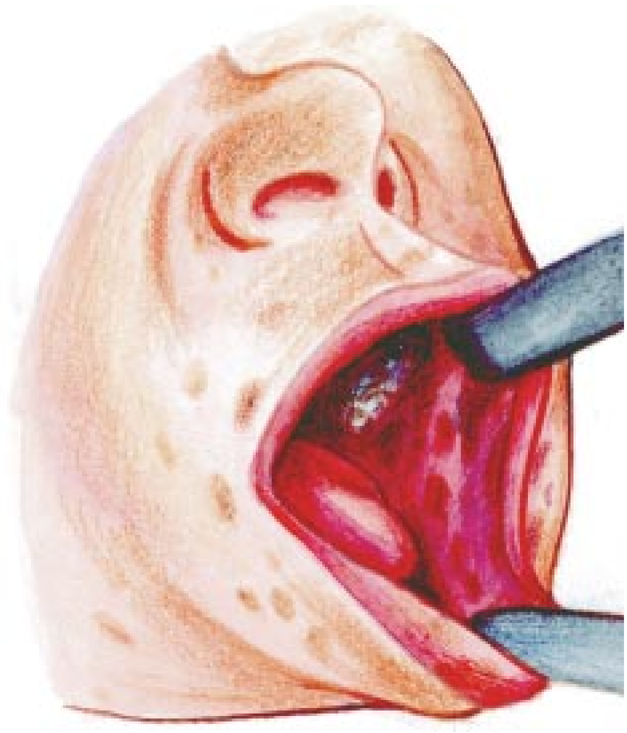


Fig. 59.11 Peutz-Jeghers syndrome.

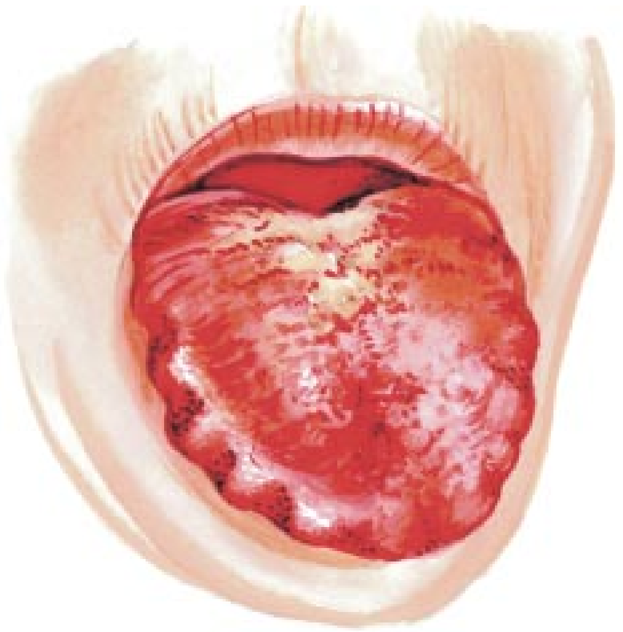


Fig. 59.12 Macroglossia (in myxedema).

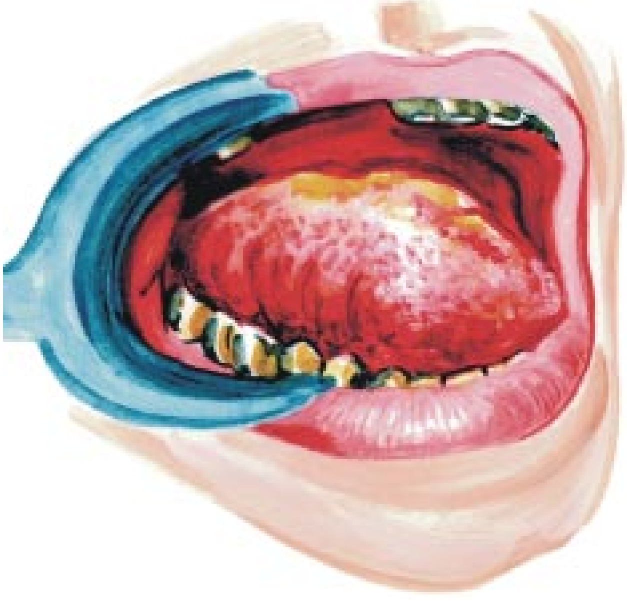
strange. When the tongue has an increased size, macroglossia, its origin can also be congenital, as in hypothyroid cretinism, or it can be acquired, as in myxedema (fig. 59.12), acromegaly, primary amyloidosis (fig. 59.13) and tumors (lingual carcinoma) (fig. 59.14).

Another alteration is leukoplakia (Fig. 59.15), which appears as a prominent, pearly, whitish plaque; They are attributed to contact with irritating factors such as tobacco and alcohol, as well as syphilis and certain forms of malignant tumor.

In addition, alterations in the color and normal characteristics of the tongue are seen, in multiple congenital or acquired processes, which constitute anomalies and true glossitis of diverse etiopathogenesis; These pathological types of language with more or less typical objective symptoms have been given proper names, true surnames of the language, considered as a name.
Let's describe the most important ones:

1. Coated tongue. It corresponds to the accumulation of filiform papillae (which stop their normal elimination process), epithelial cells, bacteria and food particles, as occurs in any pathological fasting process of any type, tongue immobility, dyspeptic processes with hypochlorhydria, stasis, constipation and digestive retroperistalsis. In contrast, subjects with hyperchlorhydria have a moist, clean, and shiny tongue. In the febrile processes and in surgical operations it is always present. The tongue coating is white or yellow, sometimes very marked; Patients refer to it by saying that they have a “ dirty tongue” and sometimes add that “they need a purgative” (fig. 59,16).
2. Peeling tongue or superficial peeling lingual swelling. The tongue appears smooth, fleshy, with the protrusions of the fungiform papillae more marked and is red, as if irritated. It is common to many infectious processes. The French point it out as the rotie tongue in typhoid fever and shigellosis. Eppinger

Fig. 59.13 Amyloid tongue.



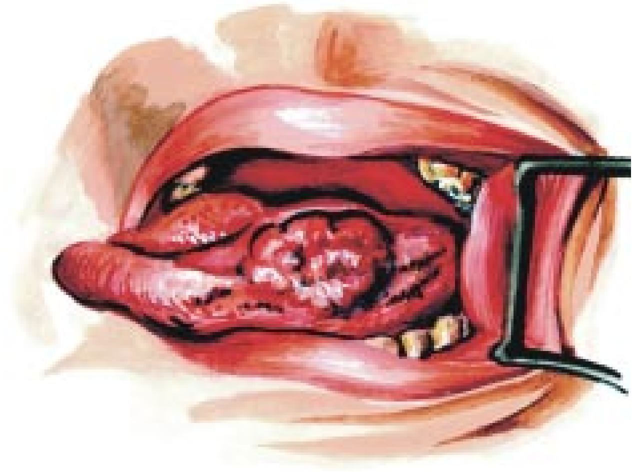


Fig. 59.14 Carcinoma of the tongue in leukoplakia.

cites a red-hot, peeling tongue in liver necrosis, associated with typical foetor oris (Fig. 59.17).

3. Geographic tongue (glossitis migrans, marginalized exfoliative glossitis or Merkel's). So called because it reminds us of a map. It is due to mixed alterations in which the filiform papillae disappear and there is proliferation and zonal hypertrophy of the fungiform papillae; These alterations form white areas that alternate with other reddish ones, in rhomboidal or circular areas, which recalls heterogeneous and variable maps over time. In reality, coated phenomena coincide with other scaly lingual inflammation. Geographic tongue is observed in chronic poisoning, chronic gastroenteritis and biliary dyskinesias. Sometimes it is family-related and dates from a very early age; It cannot be assured that there is an exact pathophysiogenic mechanism (Fig. 59.18). It evolves asymptomatically and benignly, changing aspects and location from one day to the next.

4. Black hairy tongue or melanoglossia. It is another modality poorly known in its etiology, which is characterized by presenting its base (posterior portion) very dark, almost black and covered with fine hair or well-identifiable bristles. It is noted that there is a particular hypertrophy of the filiform papillae, which become long and pigmented and undergo a cone-shaped transformation. These papillae, which become very long, are inclined "like a wheat field blown down by the wind and can be combed like hair." Its evolution is capricious and sometimes, after many years, large plaques fall off, resulting in a spontaneous cure. This type of tongue is due to the continued action of a chronic irritant agent (dyes, toothpastes, etc.) or certain fungi (Aspergillus niger) (fig. 59,19) on these papillae.

5. Fissural, scrotal or cerebriform tongue. It appears constitutional in nature and its shape resembles a leaf or the scrotum and, sometimes, the convolutions of the brain (cerebriform tongue). It has a deep central groove and multiple oblique or transverse grooves to the anterior one (fig. 59.20). It is congenital, asymptomatic and benign. Food debris accumulated in the grooves can cause glossitis.

6. Dry tongue. It is due to xerostomia and is part, along with the dryness of the buccal mucosa and gums, of the dehydration syndrome, in which it is an early symptom, which begins in the center and extends to the entire surface at the ends; Its appearance is like toast.

7. Rhombic medial glossitis. It is characterized by having a rhomboidal-shaped plate on the back of the tongue, 1 cm wide by 1.5 cm long, red in color, slightly mamelonated and smooth due to loss of papillae. It is a congenital anomaly observed almost exclusively in men; it has no semiological significance (fig. 59.21).

Different types of tongue have been described in digestive diseases, of which we will only point out a few.

In liver cirrhosis, the tongue is red, smooth, shiny, as if varnished (Pagel's tongue). The gastroduodenal ulcer has a special appearance: it is characterized by small oval-shaped, red erosions, which are located in the posterior part at the level of the midline, and which are due to the disappearance of the keratinized papillae. This tongue is called apapillosis areata (Henning).

In Plummer-Vinson syndrome, atrophic glossitis with oral rhagades appears, also common in spuriform syndromes.

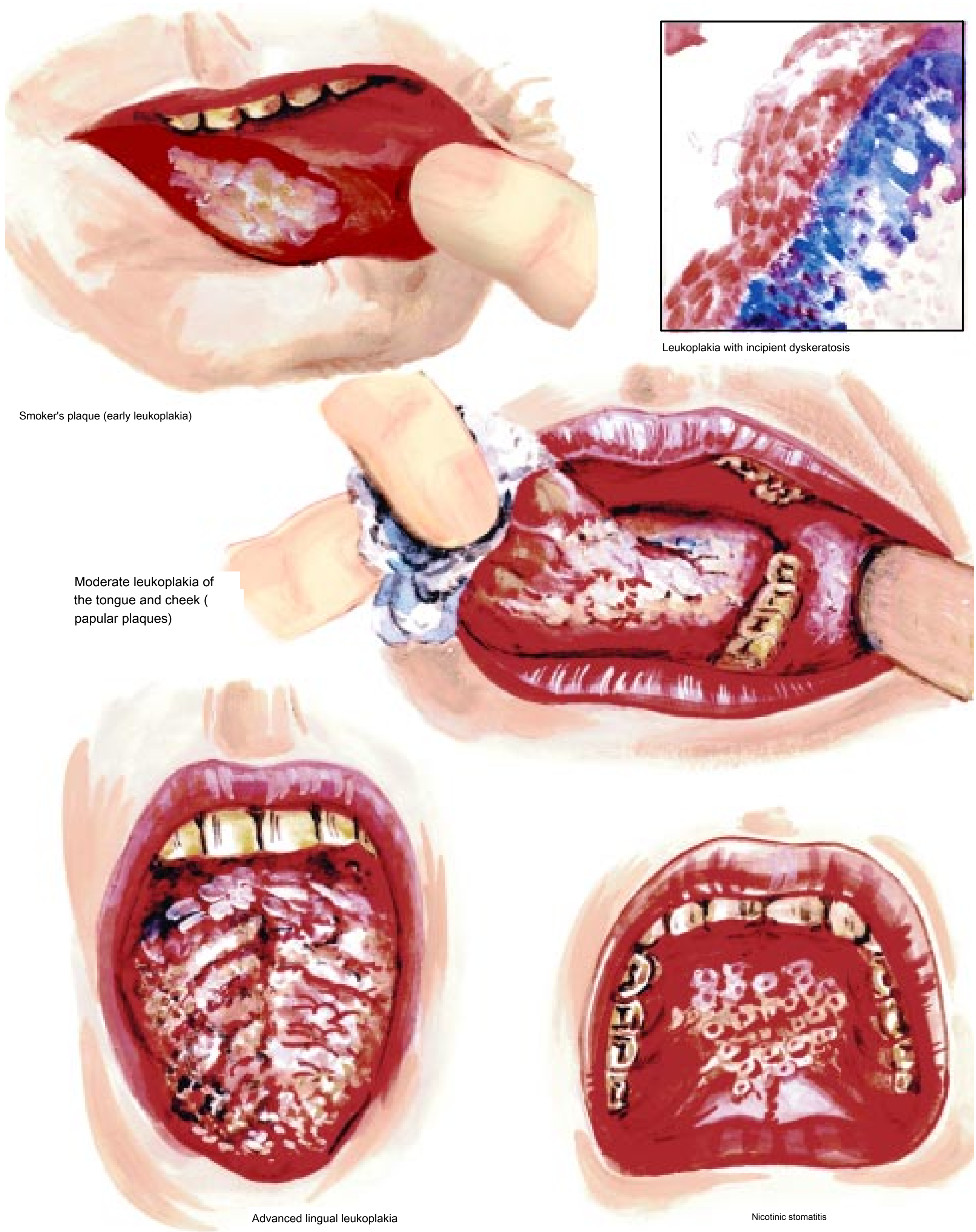
In deficiency processes and avitaninosis, the tongue of arriboflavinosis is purple-red (magenta tongue), has swollen fungiform papillae and is accompanied by cheilosis (Fig. 59. 22).

In nicotinic acid deficiency, the tongue initially turns scarlet red, dry, smooth and painful (pelagrous tongue) due to inflammation of the papillae (Fig. 59.23).

In endocrine conditions we have already pointed out the predominant macroglossia in some of them.

In hematological conditions we have Hunter's glossitis of pernicious anenmia, which presents as a depapilated, edematous, shiny and smooth tongue; This last characteristic has earned it the name varnished tongue (fig. 59.24).

In conditions of the skin, tongue, and gingival and buccal mucosa, similar lesions may occur; for example, lichen planus and others (Fig. 59.25).



Leukoplakia with incipient dyskeratosis

Smoker's plaque (early leukoplakia)

Moderate leukoplakia of the tongue and cheek (papular plaques)

Advanced lingual leukoplakia

Nicotinic stomatitis

Fig. 59.15 Leukoplakia.

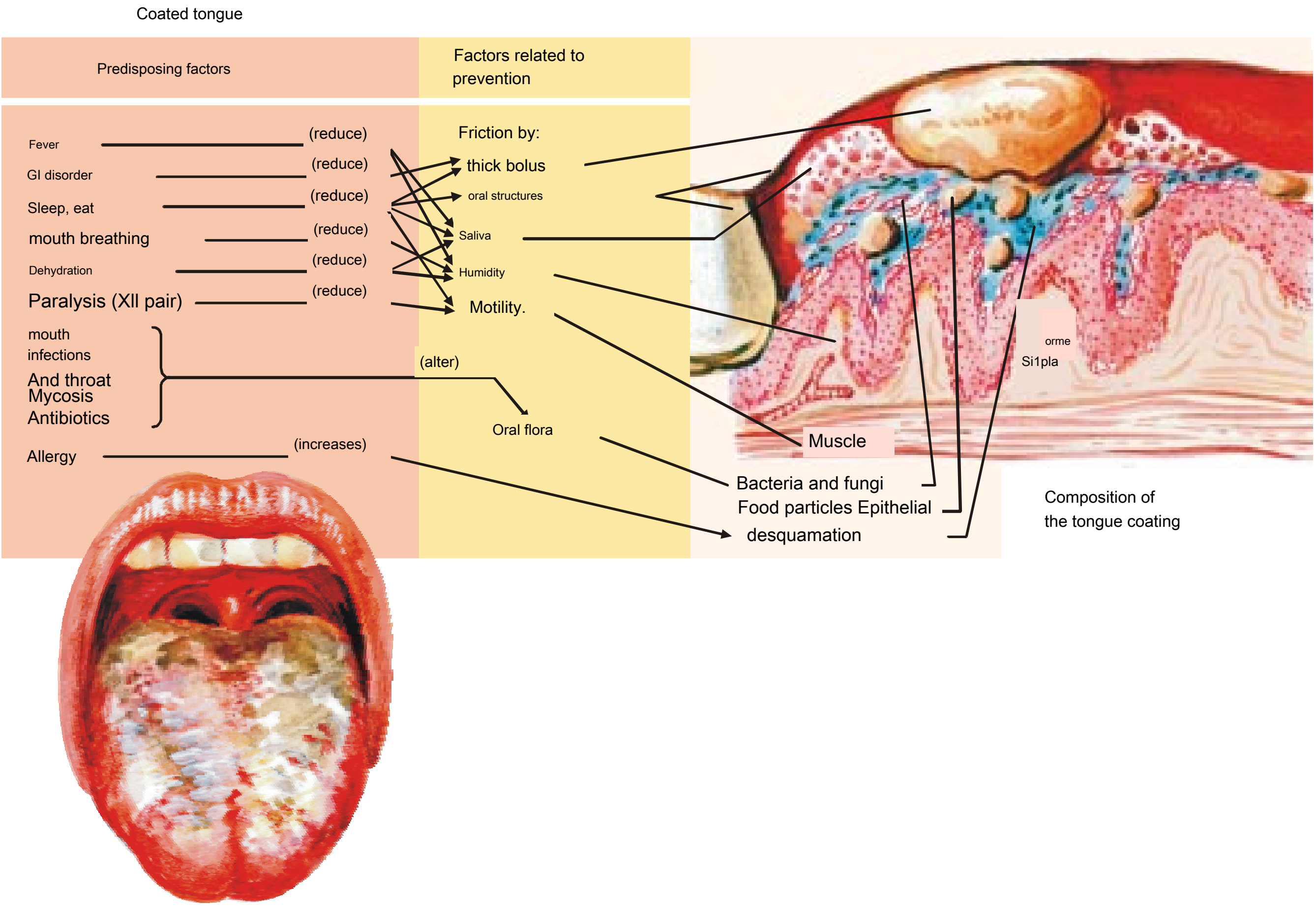

Coated tongue
Predisposing factors
Factors related to prevention
Fever
GI disorder
Sleep, eat
mouth breathing
Dehydration
Paralysis (XII pair)
(reduce)
(reduce)
(reduce)
(reduce)
(reduce)
(reduce)
mouth infections
And throat
Mycosis
Antibiotics
(alter)
Allergy
(increases)
Friction by:
thick bolus
oral structures
Saliva
Humidity
Motility.
Oral flora
orme
Si1pla
Muscle
Bacteria and fungi
Food particles Epithelial
desquamation
Composition of the tongue coating

Fig. 59.16 Coated tongue: semiogenesis and semidiagnosis.

Fig. 59.17 Flaked tongue.

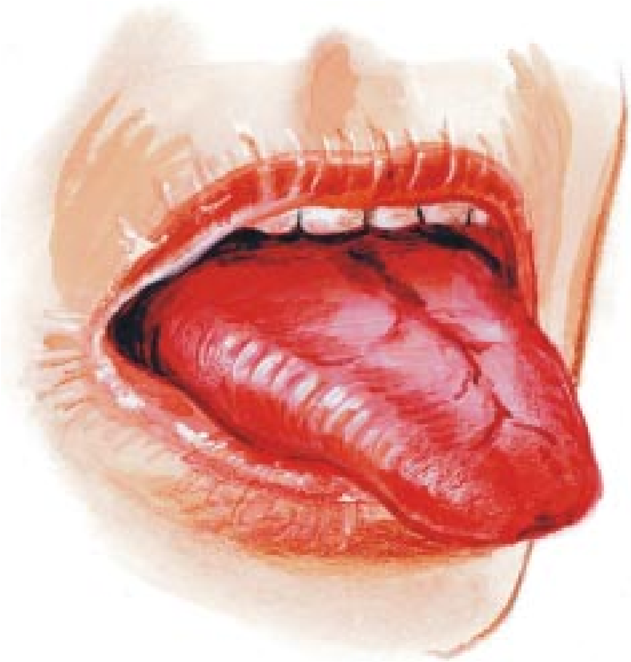
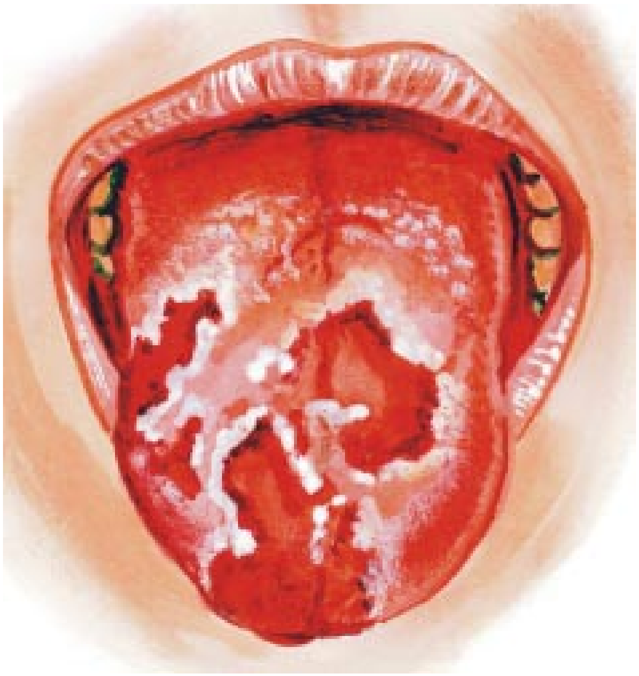


Fig. 59. 18 Geographic language.



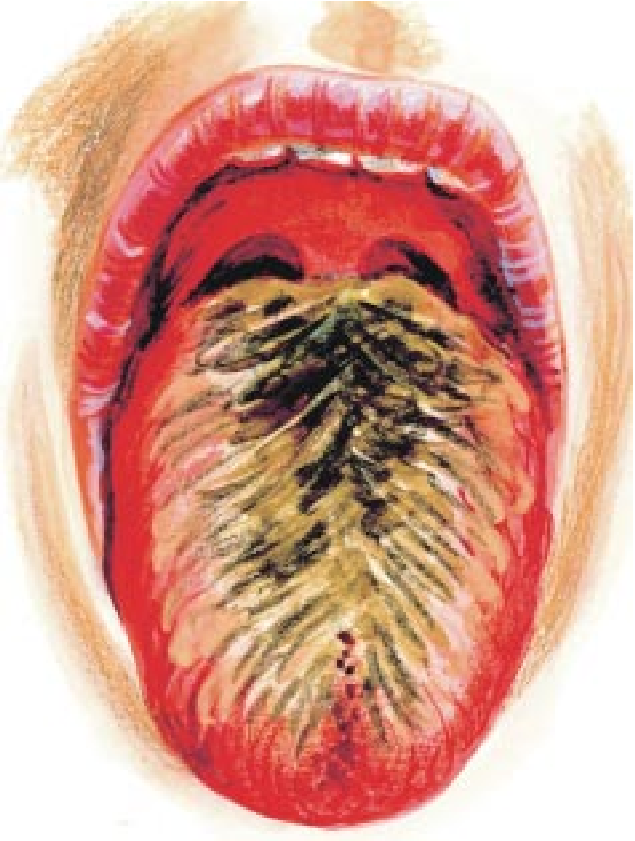


Fig. 59.19 Black hairy tongue.

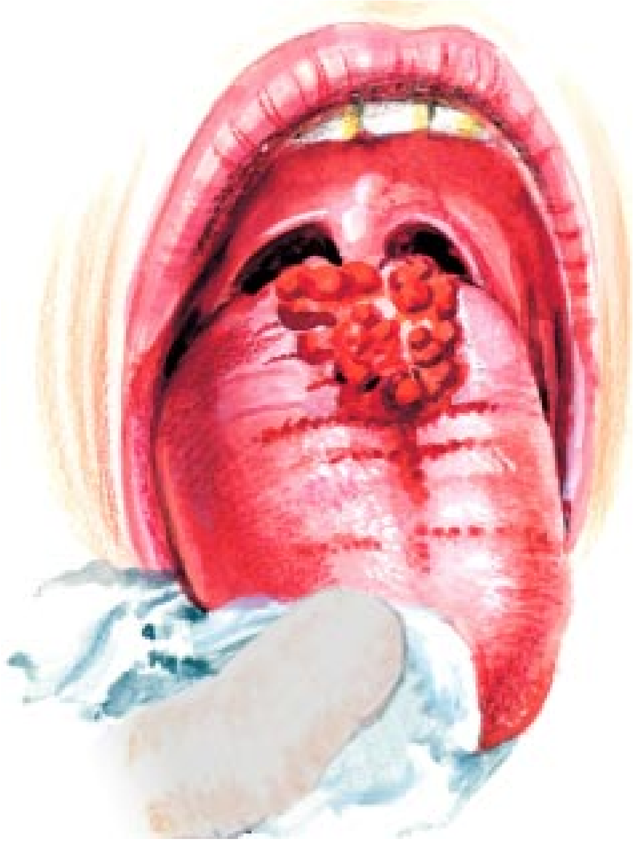


Fig. 59.21 Rhombic medial glossitis.

Fig. 59.20 Scrotal or cerebriform tongue.

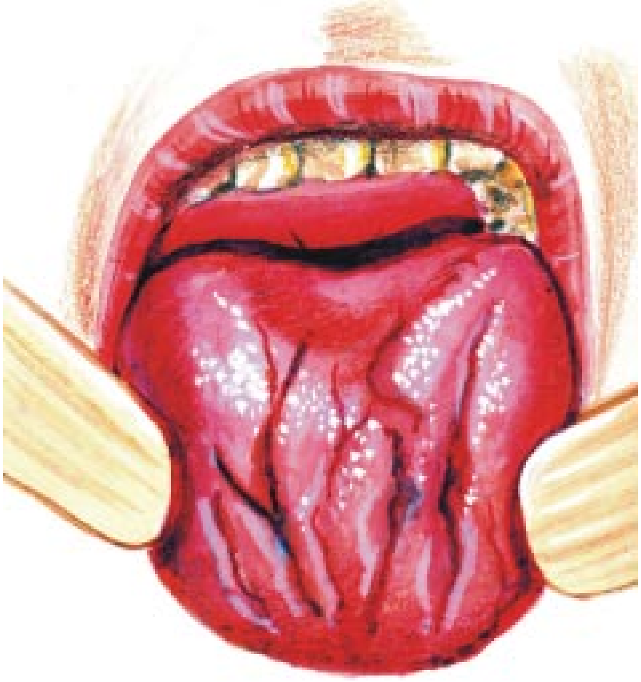
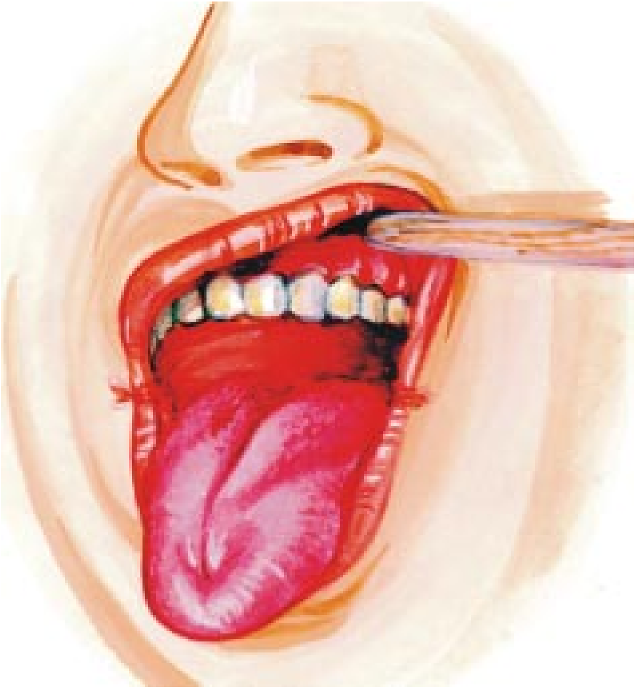


Fig. 59.22 Magenta tongue (arriboflavinosis).



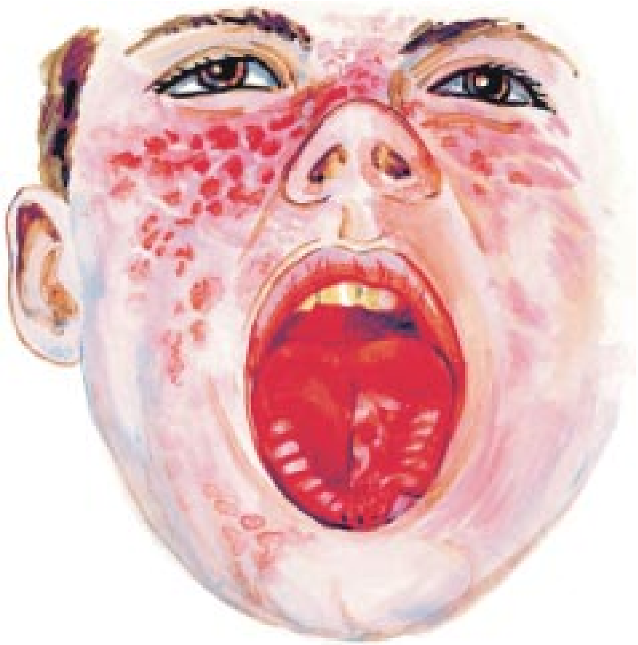
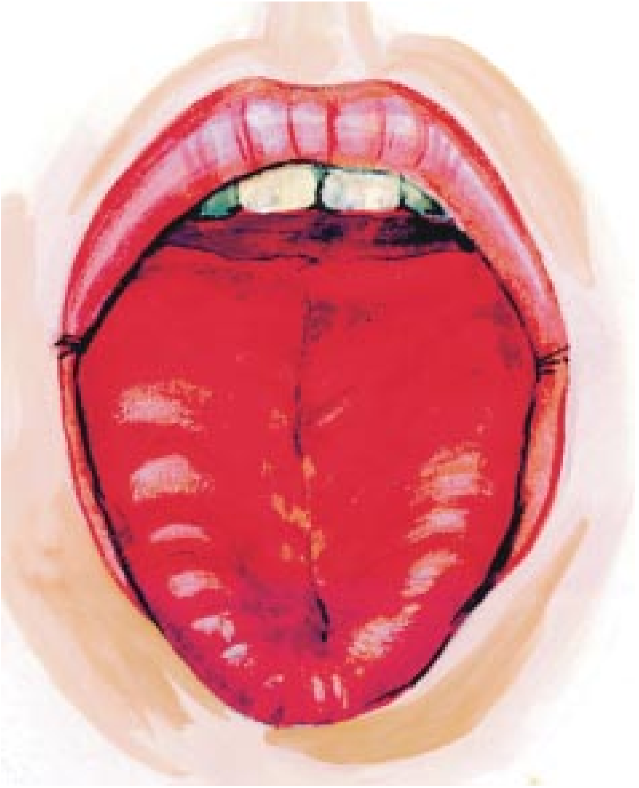


Fig. 59.23 Pelagrous tongue.

Fig. 59.24 Hunter's glossitis (pernicious anemia).



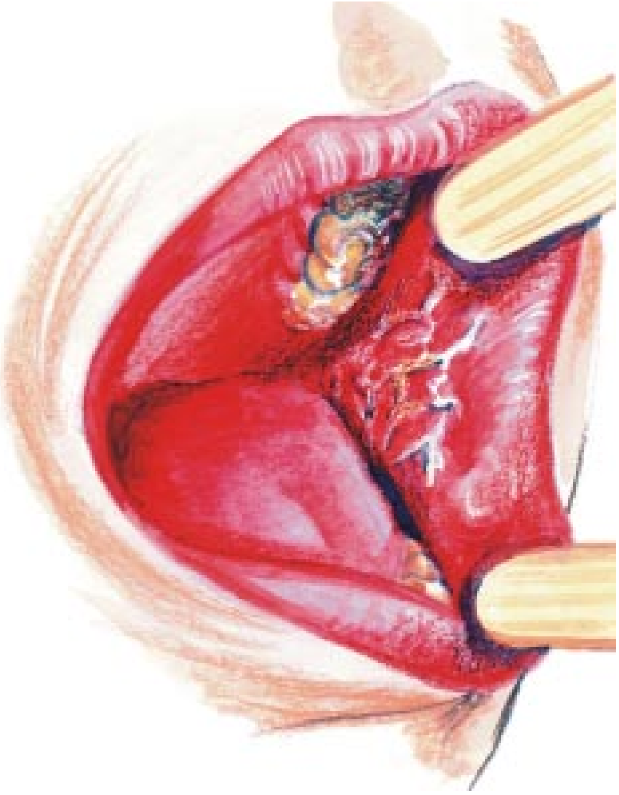
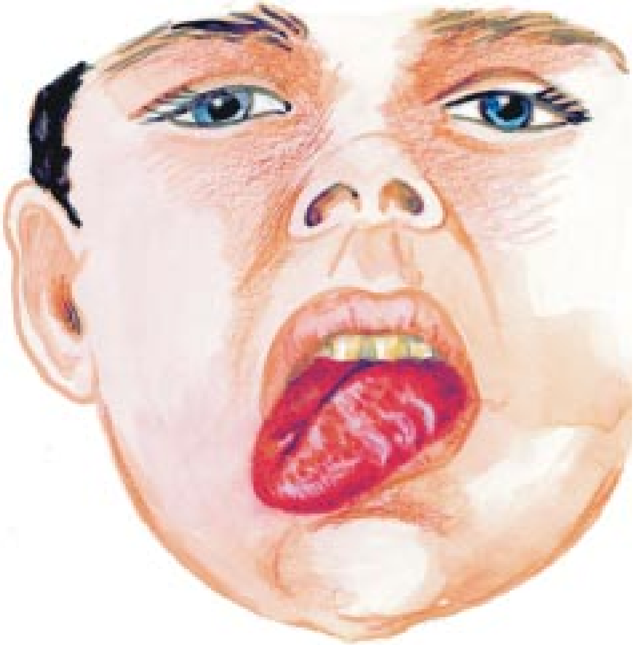


Fig. 59.25 Oral lesions in lichen planus.

Fig. 59.26 Deviation of the tongue due to hypoglossal paralysis.



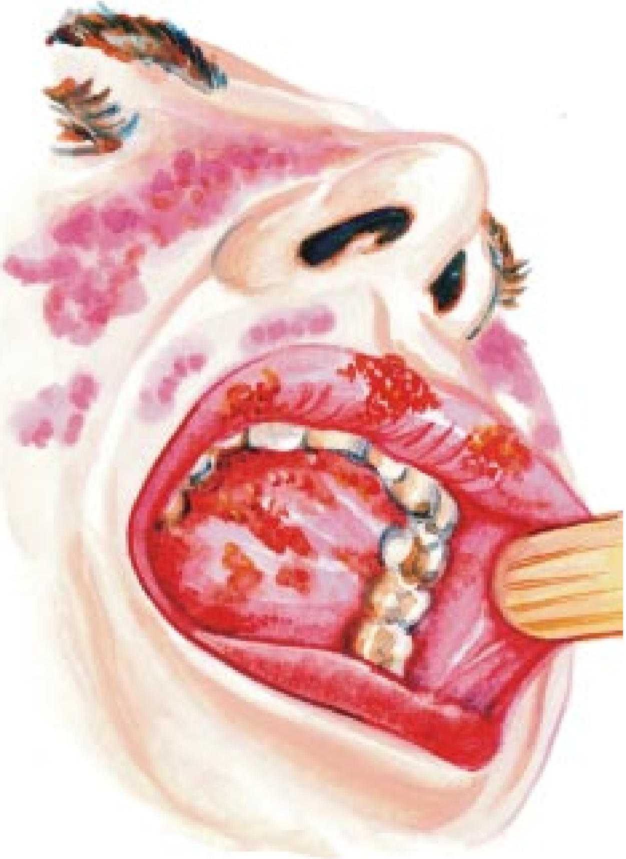
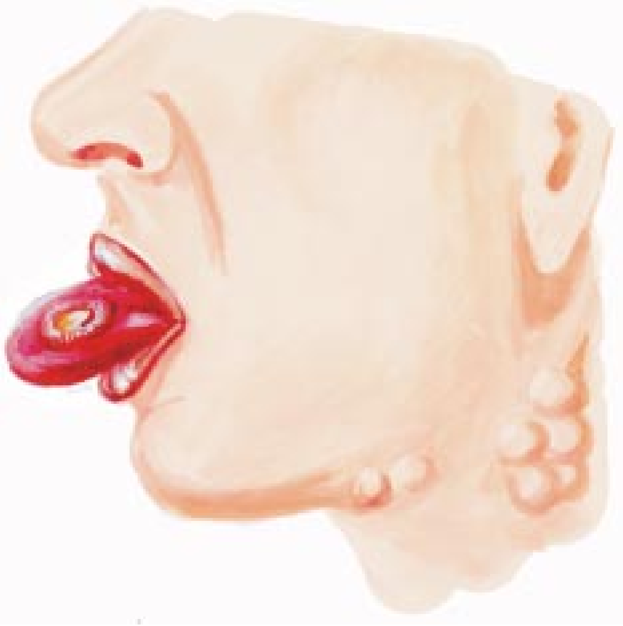


Fig. 59.27 Oral lesion in disseminated lupus erythematosus.

Fig. 59.28 Syphilitic chancre of the tongue.



In disorders of the nervous system there is paralysis or lateral deviation of the tongue in lesions that affect the hypoglossus. These conditions will be described in the semiology of that system (fig. 59.26).

In systemic conditions, such as lupus erythematosus and other collagen diseases, characteristic alterations can also be observed, especially of the buccal mucosa (Fig. 59.27).

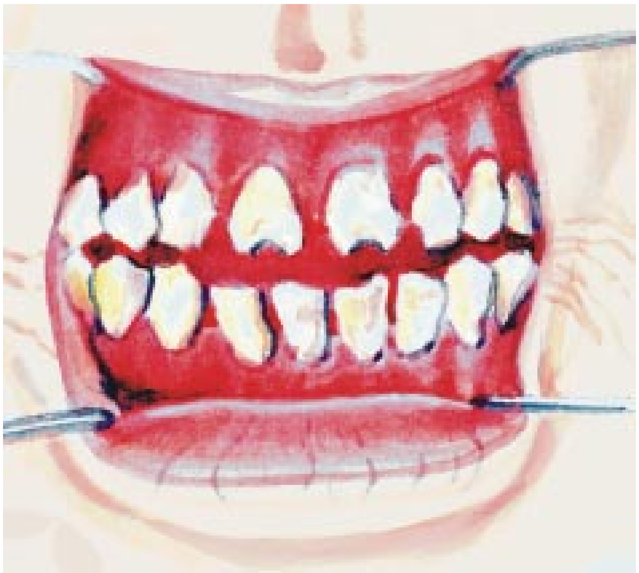
There are also ulcerations of the tongue, such as syphilitic chancre (fig. 59.28), tuberculous and neoplastic ulcerations, etc.

Teeth

Once the normal number of teeth is known, depending on the age of the patient, we can appreciate dental anomalies. Numerical anomalies, eruption (early, late), size, implantation or site, shape and structure are checked. Generally, dental anomalies are characterized by the association of several of these pathological factors. Let us cite as a typical example Hutchinson's teeth, which have been erroneously attributed only to heredosyphilis.

Hutchinson's teeth, also seen in rickets, hypothyroidism, etc., are fairly easily identified (Fig. 59.29). They present with different variants that consist of the separation of the two upper front incisors (described by Gaucher), already as a false diastema, if the frenum is interposed; The wear of the enamel forms the bevelled, screwdriver-shaped tooth, which appears wider at the base and narrower at the free edge. They also occur with an irregular or serrated free edge, with variations in size: macrodontia, microdontia or anisodontia. In exceptional cases, Hutchinson's teeth in the newborn are accompanied by otitis and

Fig. 59.29 Hutchinson's teeth.



keratitis, constituting the so-called Hutchinson triad, which does seem to depend on heredosyphilis.

Let us remember that, although it belongs to the field of the dentist, the doctor can, through simple observation, identify dental diseases: cavities, phlegmons, pulpitis, alveolar inflammation, suspicions of periodontitis due to painful percussion, as well as alterations in color and appearance, weakening, enamel wear, uncovered necks, etc., all of which is important as a possible focal infection.

Gums

They are the seat of various pathological processes: local inflammations or gingivitis, associated or not with stomatitis, and also dependent on different etiological factors, septic, deficiency (scurvy), toxic, etc. It is interesting to identify chronic lead and bismuth poisoning through the presence of bluish-black rings or lines, typical of these processes (fig. 59.30). Let us point out the possibility of tumors (epulis), fibromas and others. Finally, let us mention alveolar-dental pyorrheic gingivitis and tartaric gingivitis caused by dental tartar, which can reach pyogenic granuloma, so dangerous for proper con-

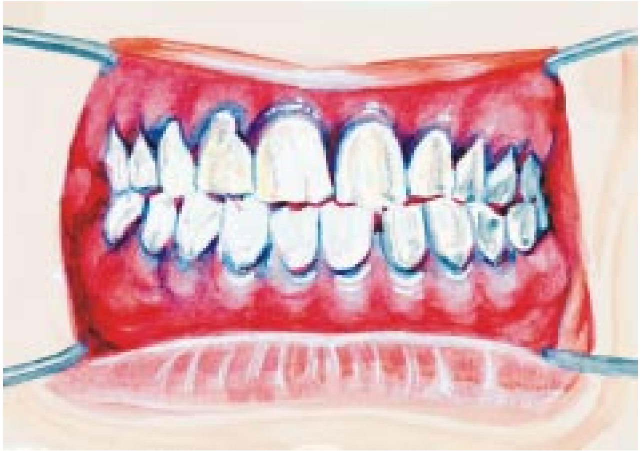


Fig. 59.30 Blue-black line in lead poisoning (saturnine halo).

Hard and soft palate

Color changes are seen, as in any mucosa, due to systemic alteration (anemia, congestion, jaundice); deformations, as in adenoid children in whom a vaulted palate appears; arched palate of heredosyphilis; benign and malignant tumors such as fibroma and congenital malformations, some of which may be of syphilitic origin (gumas). Finally, let us mention velopalatine paralysis due to injury to the internal branch of the spinal cord following diphtheria, in which cases the bell or uvula appears to hang inertly.

Salivary glands

We already knew extensively about its physiology when studying sialorrhea and xerostomia, and its clinical anatomy and semitechnics were reviewed in Chapter 16, Section I, volume 1.

In the pathological order (semiogenesis and semiography) there are multiple objective symptoms. Alterations in volume (increase) are noticeable, as are the expression of inflammatory processes, such as epidemic parotitis of viral etiology, commonly known as mumps", which is more easily observed in the parotid glands (although it can extend to the other glands, unilaterally or bilaterally). Let us also mention parotitis of another etiology, submaxilitis and sublingualitis. In addition, we must point out the increases in volume caused by salivary lithiasis with obstruction of the ducts (sialodochitis) , tumors of the salivary glands (mixed tumors of the parotid and submandibular), pansalival tumor lymphogranulomatosis or Mikulicz disease, as well as the exaggerated outflow of saliva, in a fountain, from the submandibular gland in the vasomotor phase of some cases of facial neuralgia.

PHYSICAL EXAMINATION OF THE OROPHARYNX

Although this digestive segment or middle portion of the pharynx is studied extensively in otorhinolaryngology treaties with the rest of the pharynx (nasal and laryngeal portions), in general, the frequent alterations or objective pharyngeal symptoms that are most important in daily practice are inflammatory processes, generically called "anginas" by the common people, since they are almost always associated with local pain. Angina can be red, that is, non-exudative, and is characterized by marked vascularization and diffuse erythema. They are congestive, catarrhal type pharyngitis, generally caused by viruses, as in the common cold and flu, or as another symptom of different infectious diseases. The multiple lymphatic follicles that constitute Waldeyer's ring are seen when they are infarcted in the course of tonsillitis or tonsillitis. Tonsillitis can be acute (pultaceous) or chronic with caseation (tacos). We also point out as white tonsils those caused by diphtheria, herpes, Vincent's fusospirilar association and others caused by streptococci, etc. This region of the oropharynx can be the seat of any of the processes already mentioned when talking about oral semiology (syphilis, poisoning, etc.).

PHYSICAL EXAMINATION OF THE ESOPHAGUS0

It has no value in this organ due to its deep position, which is why we use complementary exploration methods to study it.

CHANGES IN THE REGIONAL PHYSICAL EXAM OF THE ABDOMEN

60

Introduction

The abdomen contains most of the organs of the digestive system inside, hence the interest in studying this region as widely as possible in this book. However, the abdomen contains organs that belong to other systems and that will be referred to occasionally when of interest in the digestive order. This explains why the abdominal examination is not easy, much less the semiological assessment of the objective symptoms that are collected in this region.

In Chapter 5, volume I we study the techniques for exploring the abdomen as a whole, as a unit, applying the classic methods of inspection, palpation, percussion and auscultation. In this chapter we will study the pathological data obtained by each of these classic methods separately, with their great richness and multiple implications, not only in the order of their semiological significance, but also of semiogenesis and, finally, we will discuss the combined physical methods, used when it is necessary to detect certain specific abdominal alterations.

PATHOLOGICAL DATA OBTAINED BY ISOLATED PHYSICAL METHODS

We are going to describe the true essence, since the pathological data collected in the physical examination of the abdomen correspond to its objective symptoms, with which we always try, associating it with other symptoms, to reach the diagnosis. The physical or objective symptoms that are identified in inspection, palpation and percussion of the belly are multiple. The purpose of a good semiotician, and therefore of a good clinician, is to verify the truly useful symptoms.

IDENTIFIABLE SYMPTOMS ON INSPECTION

We will group them in the following way:

- Skin alterations.
- Contour alterations or pathological typology.
- Alterations of the navel.
- Volume alterations.
- Abnormal movements.

Skin disorders

Of course, the skin can present on the abdomen in all its pathology, that is, with all the elementary lesions (macules, papules, etc.), rashes, jaundice, paleness, cyanosis, etc., and other lesions, which are treated in dermatology, but which must be noted in the general physical examination. In particular, let us note the typhic roseola or lenticular spots", pink in color, which disappear with finger pressure and which are observed in typhoid fever; the angiomas or red moles, of Bouchard, selectively located on the belly and which have been considered as an indirect index of liver pathology without being able to be demonstrated; telangiectasias, of stellate shape (spiders or spider veins), located more frequently in the upper portions of the chest, shoulders and behind the ears, whose alterations point towards the diagnosis of various liver diseases, particularly cirrhosis. Let us also point out the melanodermal spots, with a similar interpretation; vergetures or atrophic stretch marks, due to the rupture of the elastic fibers of the skin (pregnancy, obesity, Cushing's syndrome, ascites, etc.); petechiae, from certain liver diseases with blood dyscrasia. These are some of the objective symptoms that deserve particular mention.

Hair distribution can also be altered, which is proven by the male distribution in women and hypertrichosis, an index of endocrine alterations, and feminization in men with advanced cirrhosis. Let us also mention the surgical scars, which suggest the pathology associated with the operation performed and perivisceritis. At the level of the scars, eventrations can be seen, which are confirmed by palpation.

Contour alterations or pathological typology

Tangential observation (fig. 60.1) allows us to specify different elements that have diagnostic utility.

Knowing the normosplanchnic or normolinear type of the abdomen, manifest deviations from the normal type can be observed, either towards macrosplanchnia or microsplanchnia. In these cases, the contour is altered, a very important fact to take into account, since it implicitly forces the semiotician to interpret the visceral projections in a different way, depending on whether the patient is a brevilinear or a longilinear patient.

The abdomen of the brevilineal or hypersthenic is prominent in relation to the thorax; It is domed and describes in its profile or contour a curve of anterior convexity that goes from the xiphoid appendage to the pubis. The anteroposterior diameters are larger than the transverse ones;
The upper portions of the belly are larger in volume than the lower ones. Charpy or xiphocostal angle is straight or obtuse. The navel appears low, as if it were approaching the pubis. They are high-bellied subjects.

It is enough to identify this type of belly in a patient to think of a numerous group of diseases such as: gallstones, pancreatitis, among others. On the other hand, when identifying this type of abdomen, one should think that the viscera are developed in the direction of the circumference and therefore, the visceral projections (the limits and particularly the painful points) will be in a relatively higher position.

The abdomen of the longilinear or hyposthenic and the asthenic is flat, excavated, small in relation to the tőrax; transverse diameters predominate over anteroposterior diameters; the navel appears high, far from the pubis, the upper portions of the abdomen appear small in relation to the lower ones; the elongated thorax;
Charpy angle is acute. These characteristics are more exaggerated in the asthenic type (Stiller's habit).

It is enough to identify this type of abdomen to think about the opposite diathesis to that of the brevilineal and, consequently, the pathology of visceral ptosis, hypotonia and atony of the digestive tract, sympathicotonias, digestive dyskinesias, etc. On the other hand, the visceral projections in this type of abdomen are expressed following the visceral development in the line, moving the limits and painful points downwards.

Navel disorders

Let us point out among the main symptoms the increase in volume of the navel determined by lumps. This increase may correspond to umbilical hernias, which are reducible or not, to benign tumors and especially to malignant tumors of the navel (primary or metastatic).

Deployment of the umbilicus and its inversion are seen in ascites; the violet-blue color corresponds to intraperitoneal hemorrhage (Cullen's sign); Deviations of the navel are observed in abdominal tumors, which push it in a certain direction, depending on the location of the lump. The " grimacing" navel (Chutro) is an alteration due to muscle contracture towards the affected side in a belly that is the seat of an inflammatory process.

Finally, ulcerated and fungal processes of the navel are sometimes observed, as occurs in infants.
We can also mention the diastasis or obvious separation of the rectus abdominis muscles in the midline, which is seen in some women with repeated pregnancies and in thin men who have good
developed these muscles.

Volume changes

As we have previously said, the normal abdomen is not prominent or excavated; Therefore, any alteration due to an increase or decrease in volume will be considered pathological. In turn, the increase or decrease in volume may be global or individual.

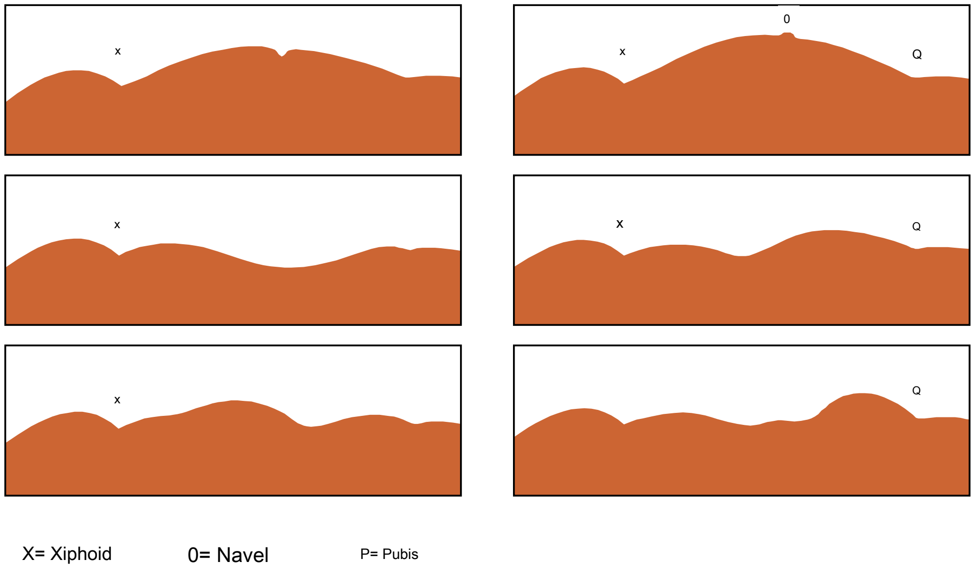


Fig. 60.1 Abdominal profiles: Careful inspection of the lateral profile of the abdomen, which may provide the first indication of abnormality, directs attention to a specific region and prompts the search for further signs.

We will place emphasis on global alterations, since those of the parcel type will be studied in the particular zonal and organ physical examination.

Let's see a summary of all these alterations and a brief explanation of them:

Global abdominal augmentations

Global increases in the abdomen correspond to obese people with large circles of fat in which the navel is hidden; They present themselves in different modalities, depending on the type of fatness, all of which corresponds to endocrine semiology. Fatty and pendulous bellies can be seen, which are known as saddle belly, apron belly, etc. In these cases they coincide with abdominal hypotonia (multiple pregnancies, etc.).

Parietal edena is observed in hydropigenic diseases such as advanced heart failure and nephrosis, for example; In the latter, the skin retains the traces of clothing at the slightest pressure.

Meteorism, at the expense of the generalized accumulation of gases within the digestive tract, globally increases the volume; Some examples are: intestinal occlusions, paresis and megacolon or Hischprung's disease. The same occurs with pneumoperitoneum, whether spontaneous or therapeutic, in which gas accumulates in the free cavity of the peritoneum (trauma, pathological perforations, etc.).

Ascites or accumulation of serous fluid in the peritoneal cavity itself increases the volume of the belly globally, with the abdomen adopting a special shape reminiscent of the belly of a frog, *belly.

- Increase of abdominal volume
  - Global (symmetric)
    - Obesity
    - Parietal edema
    - Meteorism
    - Pneumoperitoneum
    - Ascites
    - giant cysts
    - Advanced pregnancy
    - physiological phenomenon
  - Parcelarium (asymmetrical)
    - Tumors
    - Hepatomegaly
    - Splenomegaly
    - Bladder retention
    - Partial meteorism
    - Hemias and eventrations

- Decreased abdominal volume
  - Global (navicular or skiff belly)
    - Tuberculosis
    - Cancer
    - Granulomatosis
    - Cachexia
  - Parcelarial
    - Retractions
    - partial
    - Anomalies
    - rare excavations

of batrachian" with the flanks dilated. Of course, the inspection of an ascites depends on its degree, since in the gases of great ascites, the belly then becomes globular and when it is of less intensity, it can take the shape of a " howitzer" or an ostrich belly.

The navel of the ascites projects outwards and becomes erased, the patients adopt the attitude of lordosis. The skin appears smooth, white, sometimes with spots; There may be wall edema, collateral circulation, which depends on the causing disease (portal hypertension, peritoneal inflammation, etc.).

Giant ovarian cysts increase abdominal volume globally, as well as pregnancy in an advanced period (physiological phenomenon), processes that are confused in a simple inspection.

All processes capable of globally increasing the volume of the abdomen require a complete physical examination and sometimes complementary examinations for their complete diagnosis.

Plotted abdominal augmentations

Among the volume increases of the abdomen of a parcel nature, we can mention intra-abdominal tumors dependent on the different organs, as well as the nodes, vessels, etc. In particular, let us mention hepatomegaly, splenomegaly, bladder retention, which sometimes leads to confusion, organ dilations, partial meteorisms, produced by partial occlusions (Von Wahl's law), as well as hernias and eventrations. The important thing in these cases is the precision of the affected area and its characteristics, all of which we will see soon.

The global decrease in belly size is due to multiple causes; It produces an excavated, sunken abdomen, reminiscent of the shape of a boat, especially called the navicular or skiff belly, scaphoid or boat cove.

This retracted belly has very marked costal ridges and iliac crests, it is due to phenomena where retraction, muscle contracture and loss of the adipose panniculus converge. It can be observed in any cachetizing disease, particularly in many abdominal cancers, in abdominal tuberculosis and in severe inflammatory processes of the intestine, among others.

The partial reduction or retraction of the abdomen is exceptional and therefore of less semiological value, although it may occur.

abnormal movements

Respiratory movements at the abdominal level are altered in many pathological processes; They can be limited, and even disappear, due to marked abdominal contracture (peritoneal reaction) in all those abdominal inflammatory processes via reflex (visceromotor reflex) or by direct irritation of the parietal peritoneum.

Sometimes exaggerated peristaltic movements are observed, in true struggle against a limited or more or less generalized obstacle, due to vagal irritability (Kussmaul's sign), as occurs in partial or total occlusions of the hollow viscera (stomach or intestine).

Finally, vascular movements are sometimes appreciated, which correspond to the heartbeats of the vessels, and the aortic heartbeat is indicated as more relevant, either due to organic injury (aneurysms) or due to simple aortism or aortic erethismno, of a functional nature.

SYMPTOMS IDENTIFIABLE ON PALPATION

In the pathological state, palpation offers a wealth of data, some referring to possible alterations of the abdominal wall and others to the different pathological processes of the abdominal cavity.

Pathological data collected during palpation of the abdominal wall

Limited vaulting of the abdominal wall can be seen, among which we can mention the corresponding aphlegmons or inflammatory processes and tumors, generally benign, such as: lipomas, fibromas and sebaceous cysts, and malignant tumors, such as parietal lymphosarcoma of the abdomen. In general, wall tumors do not follow respiratory movements, but rather become more noticeable when the abdominal wall becomes tense (fig. 60.2). We can also verify an increase in the thickness of the belly due to edema, fat, emphysema, myxedema, as well as a decrease, which verifies what was suspected in the inspection.

Sometimes we palpate midline hernias. umbilical, hypogastric or crural and inguinal orifices, which due to their smallness may have gone unnoticed during the inspection. Sometimes they are only confirmed by coughing, when pushing, or while standing. We can verify the size, the reduction, the characteristics of the orifice and the hernial sac, etc.

Hyperalgesia or superficial caused pain is also checked, with the application of the corresponding maneuvers. This phenomenon can be observed in neuritis, radiculitis, herpes zoster, tabes, etc.

Sometimes the pain caused by the parietal region corresponds to the subcutaneous tissue or the deep muscular layers.

We will now deal with alterations in the tone of the abdominal wall.

Abdominal hypotonia is identified by palpating a soft belly, like cloth, "belly in old rag", which is seen in subjects with Stiller's habit. Muscle relaxation is observed with general malnutrition, corresponding to thin subjects, or with loss of tone, as in some women who have had multiple pregnancies, etc.

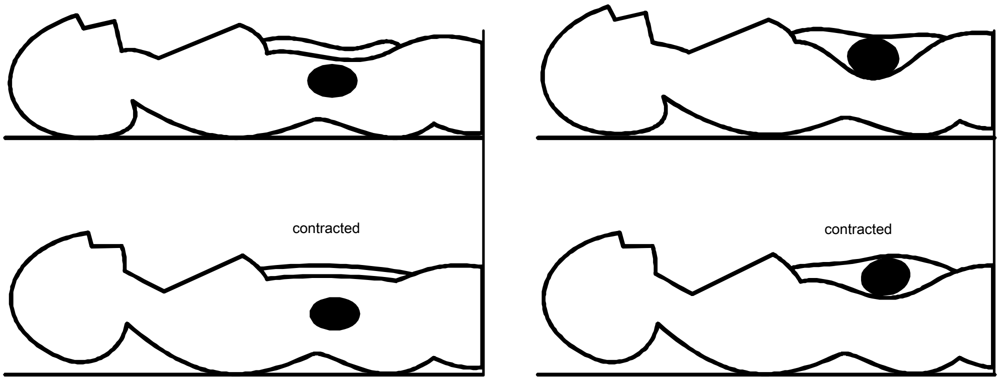


Fig. 60.2 An intra-abdominal tumor can be differentiated from a parietal (intramural) tumor by palpating while the patient raises his head: a, in the case of an intra-abdominal tumor, the palpating hand is moved away from it by contracting muscles; b, while if the tumor is parietal it is still palpable, even if the muscles are contracted.

Abdominal hypertonia has much greater semiological importance and can be due to different pathophysiological mechanisms. Let us first point out a type of constitutional hypertonia, that is, subjects that are difficult to palpate, due to marked development of their abdominal muscles or their own contracture, in which we must apply the maneuvers that we already studied in volume 1, Chapter 5, in order to achieve satisfactory palpation. Abdominal hypertonia of psychological cause is also observed in predisposed subjects, who are terrified of the physical examination, which requires great patience from the doctor. The simple contact of the hands produces "tickling" in other subjects, which creates a real problem when performing palpation.

Finally, let us mention abdominal hypertonia reflecting peritoneal processes, of great semiological value. When it is diffuse, it constitutes the so-called "board belly", an index of acute abdominal processes of a different nature. This hypertonia indicates an attack on the peritoneum by an acute inflammatory process, as occurs in acute appendicitis, cholecystitis, ulcerative perforations, rupture of an extrauterine pregnancy, among other examples. The hypertonia can be patchy, which indicates that the organ responsible for the peritoneal reaction must exist at that level.

In the navel, in addition to hernias, we can palpate tumors and granulomas, which confirms what was noted in the inspection.

Pathological data collected during palpation of the abdominal cavity (abdominal tension and viscera)

Abdominal tension is also altered by varying abdominal pressure, and depending on the degree of alteration, some more or less defined palpatory sensations can be identified that deserve special attention. They are resistance, reluctance and fluctuation.

The resistance or sensation of consistency within the peritoneal cavity corresponds to the densification of a normally hollow organ or the increase with greater solidification of a solid organ. Resistance is also perceived due to hypertension of the liquid content of the free cavity, as occurs in cases of high tension ascites. Other examples of resistance are large hepatomegaly and splenomegaly, solid tumors of the belly, etc.

Reluctance is a palpatory sensation of less resistance. In general, it corresponds to liquids or gases under tension; It is similar to the sensation obtained in cysts, hence we also speak of cystic sensation. It is easily verified in cystic tumors, in ascites at lower tension and in some cases of pneumoperitoneum, a phenomenon that is palpable like an inflated tire.

Fluctuation is the palpatory sensation corresponding to the minimum of resistance, when palpating liquid pockets at low tension as occurs in small ascites; It recalls the classic fluctuation of pus in abscesses.

These sensations of resistance, reluctance and fluctuation can logically be seen as more or less generalized or localized, depending on the nature of the process that causes them.

Let us now add some particular data from pathological palpation in relation to tumors and digestive and peritoneal collections of fluid and gas.

In the tumors, tumors or lumps of the Argentine authors, palpation must detail: shape, size, location, consistency, depth, character of its surface, presence of heartbeat, relationship with respiratory movements and with neighboring organs, presence or absence of pain, as well as the possible identification of the tissue or viscus that is the seat of the tumor, where it is

Haussmann's sliding palpatory maneuver is very useful.

Increases in the size of the viscera are also palpable. Hernias and eventrations noted in the inspection are also checked in better detail.

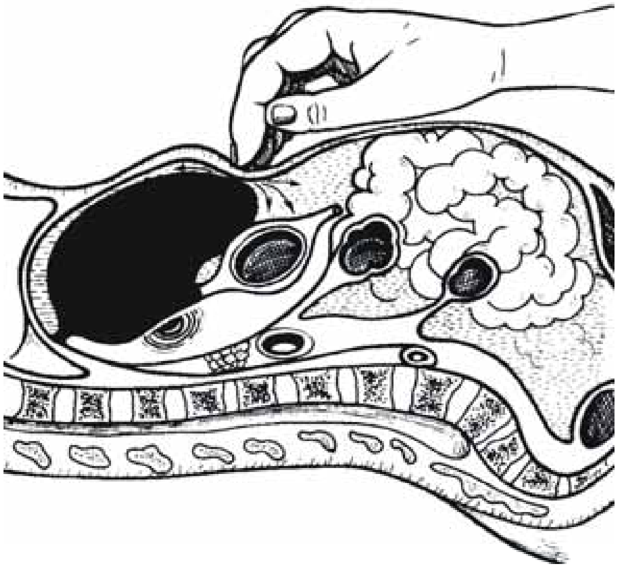
In meteorism and pneumoperitoneum, palpation is easy, since there is no hypertonia per se and this phenomenon is perceived on palpation, whether generalized or limited to a certain area.

In ascites, through pure palpation, enlarged viscera (liver, spleen) or tumors with the sensation of floating, a sign of iceberg, are sometimes identified (Fig. 60.3). Let us advance here that with percussion combined with palpation, in cases of ascites the presence of the transabdominal liquid wave of Tarral and Morgagni is verified, by palpating with one hand the wave produced by percussion on the other side of the abdomen. Sometimes you can perceive the wave as it is transmitted through the wall; When in doubt, an assistant's hand is placed in the midline along its ulnar edge and in this way we eliminate the vibration transmitted by the wall, but the true liquid wave persists (fig. 60.4).

In cases of small free ascites, it is only verified by vaginal or rectal examination, with the patient erect, as if riding a horse on our hand, observing the small bags of ascitic fluid that accumulate due to gravity in the pelvis.

We must also refer to the palpatory sensation of splashing or succussion, corresponding to the accumulation of gas and liquid in a hollow organ or segment thereof, due to phenomena of atony and obstruction.
We will return to this symptom on the occasion of the study of particular palpation by organs.

Fig. 60.3 Iceberg sign.



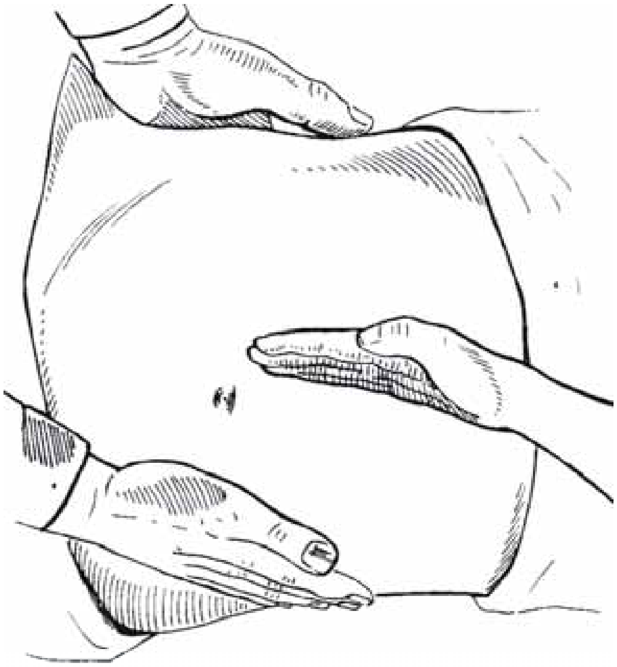


Fig. 60.4 Complementary maneuver to eliminate wall vibration.

Another important objective symptom that we collect in deep palpation and palpation of the cavity is the visceral pain itself. We already know from clinical anatomy the so-called visceral points, as well as the mechanism that produces visceral pain or Morley's peritoneovisceral reflex. Sometimes one or more larger painful points may be required, which constitute true areas of provoked pain.

Depending on the location, as we will see in the particular zonal and organ study, the presence of the symptom pain caused by deep palpation will be interpreted as corresponding to a certain affected viscus, in which there is undoubtedly a more or less marked peritoneal reaction. Pain caused in the epigastrium, for example, may be due to a gastric or duodenal ulcer or cholecystitis, depending on its topographic location. There is also provoked pain related to plexitis or irritability of the nerve plexuses of the abdominal cavity.

Let us confirm as a palpatory symptom of the abdominal cavity the vascular beats, especially of the abdominal aorta, whether of a functional or organic type, better specifying the alterations that could have been obtained in the inspection. Also important are intra-abdominal palpatory sensations of more or less localized hyperperistalsis.

IDENTIFIABLE SYMPTOMS ON PERCUSSION

In a pathological state, we must consider respectively vaguely:

Hypertympanism.
Hypotympanism.
Dullness.

These alterations of the percussive sound, in turn, can present with a more or less generalized or individual character.

The phenomenon of hypertympanism corresponds to an increase in atmospheric air or gases of another order, in the hollow viscera or in the free cavity of the peritoneum.

Hypotympanism is seen either due to high gas tension or due to a relative densification of the hollow viscera.

Dullness, in general, is due to fluids under high tension, particularly in the peritoneal cavity, to the obvious densification of a hollow viscera or to the increase in size of normally solid viscera, or also to the formation of tumor tissue at any level of the abdomen. An intestine full of excrement can accidentally be matted by percussion.

Let us detail these alterations with concrete examples in order to study the semiological significance of hypertympanism, hypotympanism and dullness.

Hypertympanism

It is observed in cases of diffuse abdominal meteorism, which can be produced either by accumulation of swallowed air or aerophagia, which causes aerogastria and aerocolia, or by the production of fermentation or putrefaction gases, or by absorption deficiency. There is also patchy meteorism, which is due to occlusion of the digestive tract at any level, in which case the phenomenon of hypertympanism is patchy following Von Wahl's law, that is, hypertympanism is found above the occluded site.

In cases of paresis or paralytic phlegmon, a very diffuse, generalized meteorism is seen, which makes the normally dull areas of the adjacent solid organs disappear. The disappearance of hepatic dullness may thus occur in these cases.

There is also hypertympanism in cases of pneumoperitoneum or free gas in the peritoneal cavity (without reaching great tension), whether traumatic or therapeutic; The disappearance of hepatic dullness is also observed, which is a sign of great value.

In partial hypertympanism, the limits of percussion of the solid organs are altered, an aspect that we will study in the next chapter, of the particular physical examination of the abdomen by Zones and organs.

Hypotympanism

High tension pneumoperitoneum is observed in some cases.

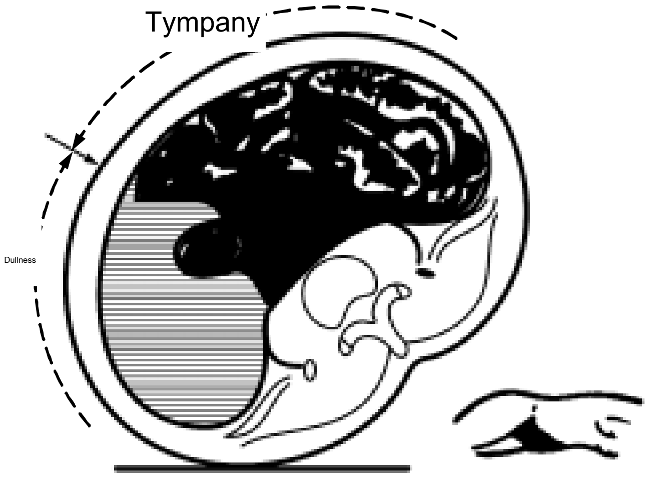
Dullness

It is observed in free ascites, also called water dullness; In it, the peritoneal dullness adopts a special geometric distribution in the belly, describing in the upper part a curved line of concavity upwards, since in the lying position (supine position) the liquid rises more on the flanks than in the center, in which place the intestinal loops float giving a tympanic sound. As the patient changes position, the fluid in the so-called free ascites tends to move and therefore the percussion lines vary, moving with the fluid the matte and tympanic sounds, respectively. Standing, due to gravity, the liquid descends, which is why there is dullness in the lower parts and tympany in the upper part. In lateral decubitus the sound is inverted, becoming dull on the side on which the patient lies, and tympanic on the opposite side, where the intestine floats (fig. 60.5).

In cases of septate ascites, in which the fibrous tissue " encysts", so to speak, liquid portions, as seen in certain inflammations - tuberculosis, for example - the dullness is seen in the liquid sacs alternating with the areas of intestine, in which there will be tympany (fig. 60.6). Sometimes the partitions are multiple and permanent, to give rise to the percutionary figure known as a " checkerboard."

Let us point out the exaggerated dullness that we obtain in the hypertrophy of solid organs, the dullness of solid tumors of a different nature and that corresponding to hollow viscera densified by filtration or tumors. In good-sized cysts, as in ascites, dullness may be observed, but in them it will be more or less fixed. Furthermore, in large ovarian cysts the dullness occupies the central region of the abdomen, because the loops move towards the flanks, the opposite of what happens in ascites. This simple maneuver allows the differential diagnosis to be made.

Fig. 60.5 Patient in left lateral decubitus position: free ascites, displacement of percussive areas.



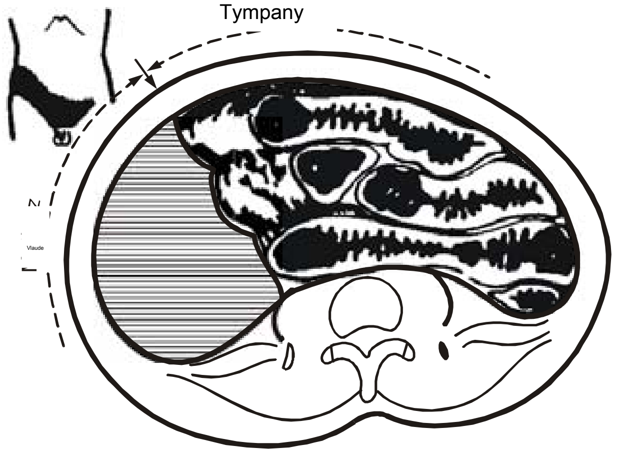


Fig. 60.6 Percussion in septated ascites: fixed areas of percussion.

SYMPTOMS IDENTIFIABLE ON AUSCULTATION

With auscultation, pathological intestinal sounds (hydroaerial), vascular sounds, and rubbing of the abdomen are identified.

The detection of hyperactive and increased frequency air-fluid sounds is of great help, along with other general, abdominal and digestive system symptoms, in the diagnosis of mechanical flounder, due to intestinal obstruction (struggle sign), which are produced by increasing peristalsis above the obstruction, in an effort to try to overcome the obstacle to intestinal transit; while absent or hypoactive fluid sounds help in the diagnosis of reflex paralytic ileus, due to many non-obstructive causes, including disorders of fluid and electrolyte balance, mainly dehydration and hypokalemia. More rarely, the absence or marked decrease in air-fluid sounds is an expression of the very advanced phase of an intestinal obstruction, when the neuromotor fighting mechanisms are exhausted.

Auscultation of abdominal friction (inflammation of the hepatic serosa, splenitis and perisplenitis, as examples), are of less practical importance in digestive semiology and vascular sounds audible in the abdomen are usually due to alterations not directly related to the digestive system.

COMBINED PHYSICAL METHODS IN ABDOMINAL SEMIOTECHNICS

In abdomen semitechnics, a combination of classic exploration methods is sometimes performed.
As the most important, we mention the combined methods for detecting ascites and determining collateral venous circulation.

EXPLORATION OF FREE ASCITIS

As already mentioned, combined palpation and percussion to explore the ascitic wave is a method known as the Tarral or Morgagni maneuver.

It is performed by striking two or three strokes with the balls of the fingers of one hand, hitting one of the flanks and palpating the opposite side with the other hand, to perceive the surge of free ascitic fluid in the peritoneal cavity; It is a liquid wave transmission phenomenon (fig. 60.7). When it is suspected that the vibration of the abdominal wall and not the liquid wave is being transmitted, the maneuver is completed by placing the hand of an assistant, along its ulnar edge, in the midline, to immobilize the wall (see Fig. 60.4).

There is also a combination of percussion and auscultation to perceive the ascitic wave, as well as the percussion of a coin on another on one side of the belly while auscultating the other side.

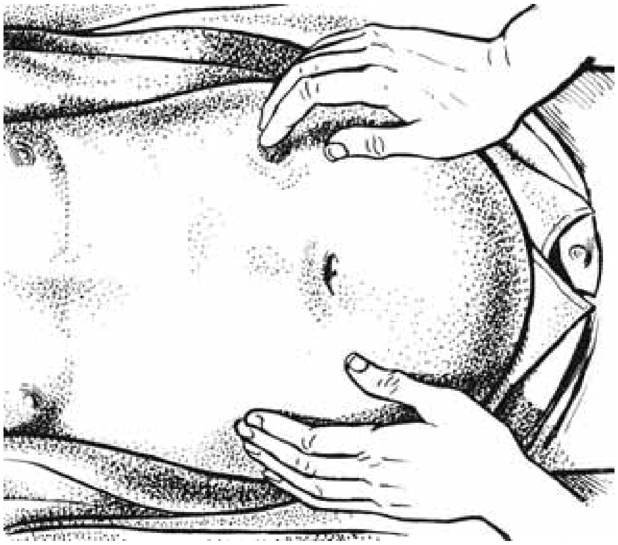
Finally, the auscultation of the voice, transmitted through the abdominal cavity filled with gas, is carried out, making the patient pronounce the word * thirty-three".

These last two combined maneuvers are similar to those used in semitechnics of the respiratory system, although their application to the abdomen is very limited.

COLLATERAL VENOUS CIRCULATION

The combination of inspection and palpation to determine collateral venous circulation has already been studied in Chapter 25, volume 1.

Fig. 60.7 Combination of palpation and percussion to explore the ascites wave.



PARTICULAR PHYSICAL EXAMINATION OF THE ABDOMEN BY ZONES AND ORGANS

61

Introduction

Those of us who dedicate ourselves to pedagogical work know that exact didactics is sometimes impossible.

This chapter could theoretically be developed in two independent parts, but it would fall into multiple sterile repetitions, which is why we have decided to do it in one go, simultaneously analyzing the different areas of the abdomen with the different organs that predominantly correspond to each particular area. We know that the correspondence is not absolute, since no area contains a single digestive organ, nor vice versa, but, in general, said correspondence is relative enough to be able to do it advantageously in a simultaneous manner0, a criterion that brings us closer to the way in which the facts are presented in daily clinical practice.

EXAMINATION OF THE STOMACH AND EPIGASTRIUM

CLINICAL ANATOMY

As particular data, we will point out that the stomach projects in its eight ninth parts to the left of the midline. The fundus or greater tuberosity reaches the diaphragmatic vault, corresponding behind the upper edge of the eighth rib at the level of the tip of the scapula (D8) and in front, at the level of the fourth left intercostal space inside the manillar line. The cardia is located behind, to the left of D10 (lower than the greater tuberosity) and in front at the level of the sixth or seventh left intercostal space. The pylorus projects posteriorly at the level of LI and anteriorly at the point of intersection of the right line with the angle of union of the seventh and eighth cartilages.

The lowest portion of the stomach is located behind, at the level of L3 or L4, and in front, at the level of about 3 cm (three finger widths) above the navel.

Let us particularly describe the Traubbe and Labbé areas (fig. 61.1).

Traubbe's zone appears projected in the lower portion of the left rib cage like a crescent, and its limits are: on the right, the left lobe of the liver; on the left, the spleen and pleural cul-de-sac; upwards, the heart; and downwards, the left costal margin.

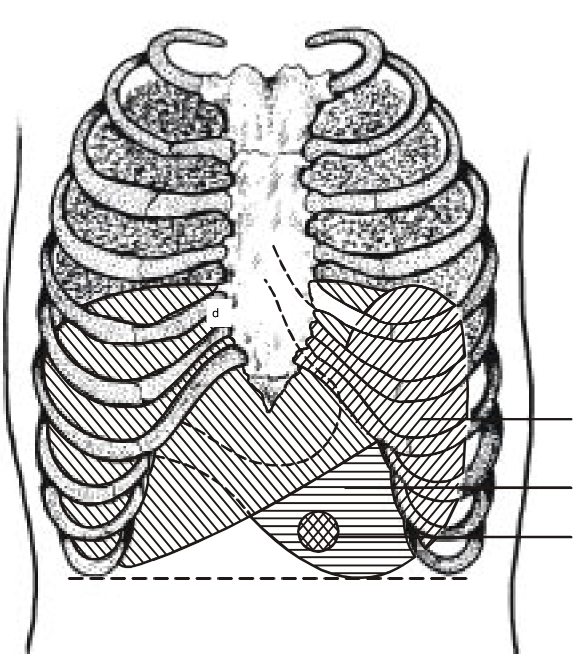


Fig. 61.1 Traubbe's semilunar space (a). Labbé triangle (b). Mendel's painful zone (c).

Labbé's zone is triangular in shape, the right side of the triangle corresponds to the lower edge of the liver, the left side to the left costal margin, and the lower side to a conventional horizontal line that passes through the free edge of both costal margins. Within this triangle a more limited circular area of the stomach is also described, which is known as Mendel's painful area.

SEMIOTECHNICS

We will refer only to the maneuvers used in the examination of the epigastric area and especially the stomach and duodenum. Superficial and deep monomanual palpation will be the most used. As always, the abdominal wall will be palpated first and then its contents in said area. The stomach, as we have seen, offers an upper portion beyond the reach of palpation because it is protected by the thorax (Traubbe's zone) and a palpable lower abdominal portion (Labbe's zone). With particular palpation of the epigastrium, possible gastric bazuqueo or Chaumel splash is explored, using small palpatory movements.

Sometimes one hand is placed on top of the other for the same purpose, pressed deeply and the hands are raised, alternating, giving rise to the classic sound of liquid and retained air (fig. 61. 2). With the pads, possible painful points in the region are looked for, as well as other physical alterations. There are the Glenard and Leven maneuvers, applicable in the case of epigastric pain due to possible ptosis that we will describe when studying epigastric pain.

Fig. 61.2 Exploration of gastric bazuqueo.

NORMAL DATA

On inspection, nothing is seen in the healthy subject. On palpation, in thin subjects, the greater curvature is exceptionally appreciated, about 5 or 6 cm above the navel, through sliding palpation, using two or three fingers (see Fig. 5. 15, volume 1, Section I). The pylorus, in 20% of normal cases, can be palpated as a cylinder about 2 cm long, in a localized area, above and to the right of the navel. On percussion, Traubbe's semilunar space, already described, is identified, which is usually tympanic (air chamber of the stomach).

PATHOLOGICAL DATA

In the inspection we must consider volume alterations and abnormal movements.

In volume alterations, we point out the vaulting of the epigastric area, which generally corresponds to tumors or lumps, predominantly of the stomach, due to infiltration of the organ, or to dilations due to pyloric obstruction, which can appear temporarily (phantom tumors) or permanently. Let us also point out eventrations and epigastric hernias. On the contrary, depressions limited to the epigastrium are observed, due to parietal retraction, in some tumors, perivisceritis and marked loss of the adipose panniculus; In addition, midline depression is observed due to diastasis or separation of the rectus abdominis major.

In abnormal movements, let us first point out the peristaltic alterations of the region, of great semiological value in the diagnosis of organic stenosis of the pylorus. In these cases, particularly in large dilations with fighting hyperperistalsis, the presence of waves that are directed from top to bottom and from left to right is observed on inspection, which correspond to pathological peristaltic movements that go from the cardia to the pylorus. The peristaltic agitation itself, with visible and moving waves, is described by the name Kussmaul sign. The Bouveret or Cruvelhier sign (stenosing ulcers of the pylorus) describes a visible cylinder with the shape of the stomach, relatively static, corresponding to the mass contracture or permanent wave of the organ in struggle.

Secondly, let us point out the abnormal movements corresponding to pathological vascular beats in the region. They are due to right ventricular hypertrophy or abdominal aortopathies, whether organic or functional. Particularly, the functional type (aortic erethism) is observed in vegetative dystonias with hypersympathicotonia, and is the objective equivalent of the "father" or "mother's illness" described in the anamnesis as a subjective and observable symptom in pythiaic women and men.

During palpation we must study the pathological data corresponding to the wall and the cavity, at said epigastric level, especially referring to the stomach.

The first pathological finding may be spasticity or parietal contracture, which fundamentally indicates the presence of acute epigastric processes with peritoneal reaction. A perforated ulcer, cholecystopathy, acute pancreatitis will be preferred examples. Let us point out other parietal alterations, such as induced pain and non-cavitary tumors.

Gastric bazuqueo, known as splashing or glu-glu noise, is also appreciated. Palpation can also be done with the fingers, as if to telegraph, causing said hydro-aerial noise, which corresponds to cases of gastric retention due to obstructive pyloric syndrome. This phenomenon implies the decrease or absence of tone and peristalsis. Gastric bulging is reported in normal subjects in exceptional cases, after having eaten.

Stomach tumors are accessible to palpation, in special circumstances of size and location. Tumors of the body and pyloric antrum should be mentioned in particular. We must specify their shape, sensitivity, mobility, relationship with respiratory movements, whether they are pulsatile, etc. Tumors originating in other viscera can also be palpated in the epigastric area: liver, gallbladder, pancreas, transverse colon, diaphragm, as well as in vessels and lymph nodes, which present with their own symptoms and characteristics.

The pain caused by the epigastric region has great semiological value (peritoneosensitivity reflex, Morley), and is common in ulcers of the stomach and duodenum, as well as in solaritis. The location is variable and multiple, since according to Head's metameric distribution, it will vary according to the exact location of the production process.

Let us mention the positivity of the Glenard and Leven maneuvers (Fig. 61.3) in the provoked pain observed in solaritis due to gastric ptosis.

In the Glenard maneuver, the doctor stands behind the patient, takes his stomach in his hands and suspends it upward, the patient experiencing a sensation of relief, which is replaced by pain when the doctor releases his abdomen. This phenomenon is known as the Glenard girdle sign.

The Leven maneuver consists of causing solar pain with the right hand (the index finger, especially) on the epigastrium, and with the left hand using the ulnar edge, the viscera is lifted and the pain is attenuated as the stomach is raised. It is known as Leven's pain sign.

Other processes such as peristaltic alterations and vascular beats, which have been described in the inspection, and eventrations and hernias of the epigastric region, are also verified on palpation.

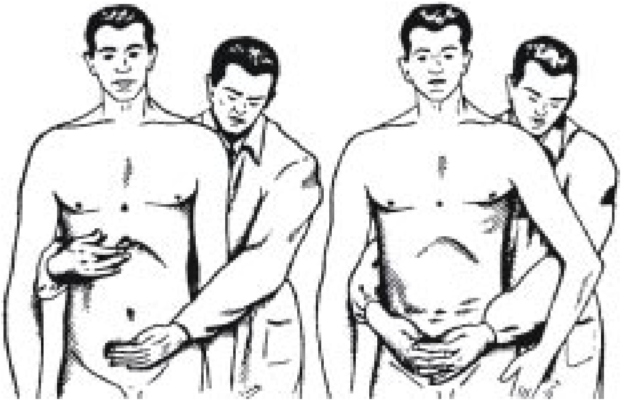


Fig. 61.3 Leven maneuver (pain-cue sign) (a).
Glenard maneuver (b).

EXAMINATION OF THE LIVER, GALLBLADDER, PANCREAS AND RIGHT HYPOCHONDRIUM

Although part of the liver, the gallbladder and the pancreas project into the epigastrium, we have preferred to carry out its particular physical study simultaneously with the examination of the right hypochondrium - where the anatomical projection of some organs is predominant - due to the repercussion that the pancreatic processes have on the liver, especially those located in the head of this organ.

CLINICAL ANATOMY

Liver

Its convex or superior portion corresponds in front to the fifth costal cartilage at the level of the midclavicular line and to the VI rib at the level of the axillary line.

The lower edge is located behind the column vertebral at the level of the XII rib, it touches the axillary line in the

In the macrosplanchnic type, the lower border of the liver is higher and horizontal, it is completely hidden behind the right costal margin, which makes normal palpation difficult. In the epigastrium it becomes more accessible as it follows its horizontal course to the left. In the microsplanchnic type, said lower edge is oblique and ascends from right to left, protruding slightly on the right costal margin, then directed to the epigastrium, where it becomes less palpable (fig. 61.4). Of course, in cases of anomalies all this varies, gaining semiological value. When the subject is standing, the liver descends in its projections.

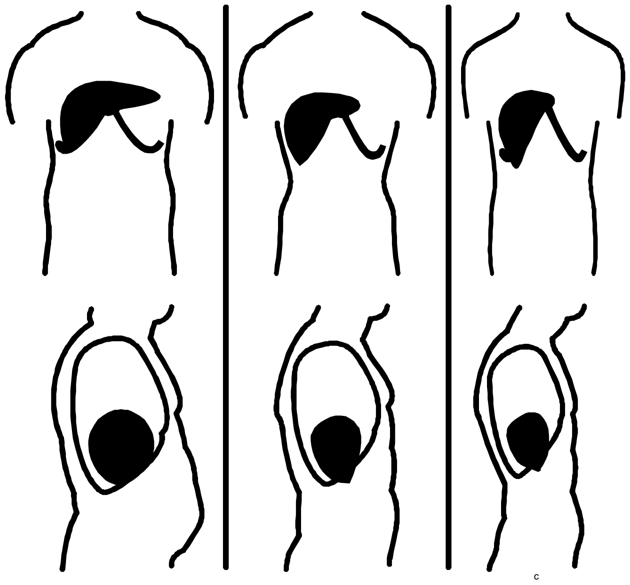


Fig. 61.4 Frontal and lateral projection of the liver: a, in the macroplanchnic; b, in the normosplanchnic; c, in the microsplanchnicus.

Gallbladder

It is practically embedded in the lower surface of the liver, it is only explorable at its bottom, which projects at the lower edge of the liver, at the level of the X rib at the point of intersection with the right edge of the rectus abdominis major. Its major axis varies: standing, parallel to the spine, and lying down, more parallel to the edge. The common bile duct projects into the area of the head of the pancreas, which we will describe below.

Pancreas

This organ projects posteriorly at the level of Ll or L2 and anteriorly at the intersection of the midline with the horizontal that joins the VII or VII rib on each side.

The head projects particularly in the pancreaticocholedociana zone, of Chauffardy Rivet, a triangular zone formed by the supraumbilical midline, a line that goes from the navel to the right axilla and the costal margin (fig. 61.5).

The body and tail extend to the left, into the epigastrium and left hypochondrium.

SEMIOTECHNICS

Palpation of the liver, gallbladder and pancreas, and percussion of the liver are studied in particular.

Palpation of the liver

In palpation of this organ we use different maneuvers:

- Simple or monomanual palpation.
- Bimanual palpation of Chauffard or rally.
- Gilbert's bimanual palpation.
- Mathieu bimanual palpation.

Palpation of Devotee.

Glenard bimanual palpation (thumb palpation).

Simple or monomanual palpation

With it, an overall impression of the liver and gallbladder is obtained; it is made with the doctor's right hand (located to the right of the patient), below the costal margin. The hand is placed flat, parallel to the edge and the patient is invited to breathe deeply.

Fingers can be used to delimit the lower border, as well as to maneuver the iceberg in cases of ascites. With this palpation we get the impression of the size, surface and consistency of the organ (fig. 61.6).

Bimanual Chauffard or rally palpation

It consists of handling both hands, the left behind, on the right lumbar region, and the right hand in front, obtaining an impression of the liver passing through the kidney (fig. 61.7). Without a doubt, it also allows us to obtain an impression of the edge and surface, as well as the consistency of the organ.

Gilbert's bimanual palpation

In its first stage, it is used to explore the anteroinferior edge of the liver; it consists of capturing said edge by tracing the right hemiabdomen with both hands from bottom to top and vice versa. The right hand is placed at right angles to the left, both touching at their free ends (fingers) (fig. 61.8). The second time to explore the posterior border is of exceptional use.

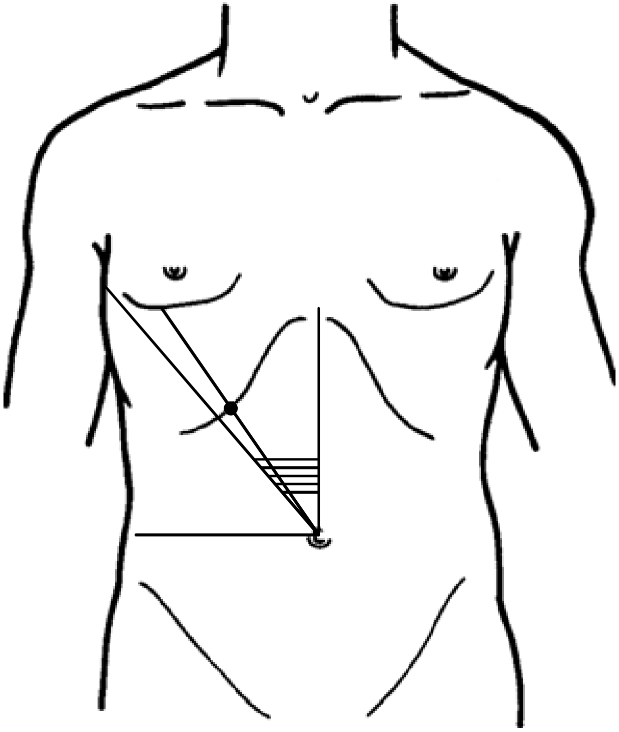
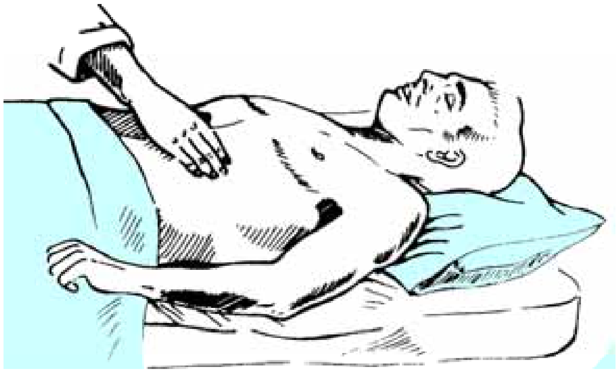


Fig. 61.5 Pancreaticocholedochal zone of Chauffard and Rivet (a). Cystic point (b).

Mathieu's bimanual palpation

We proceed as if the doctor were going to palpate his own liver. The two hands, in contact with the index fingers, are placed to the right of the navel on the abdominal wall and are traced from bottom to top until reaching the lower edge of the liver as if trying to pass under it. The respiratory modality is also performed, leaving the hands fixed at the level of the liver and it is expected that this organ will come into contact with the fingers during inspiration (fig. 61.9).

Fig. 61.6 Single-manual palpation of the liver.



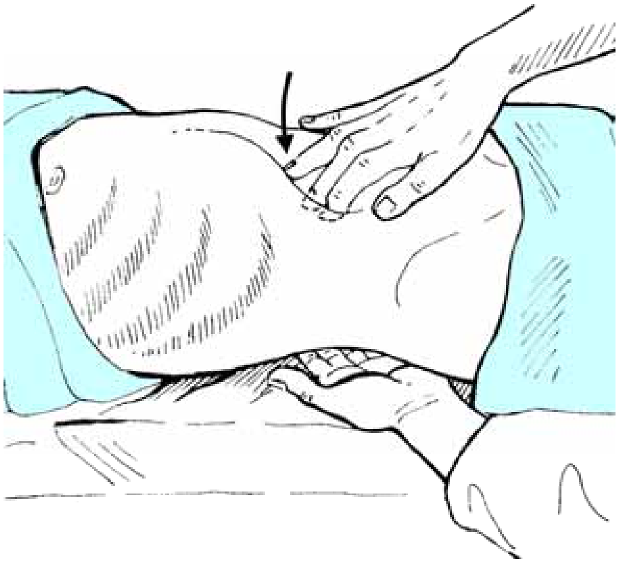


Fig. 61.7 Bimanual Chauffard or rally palpation.

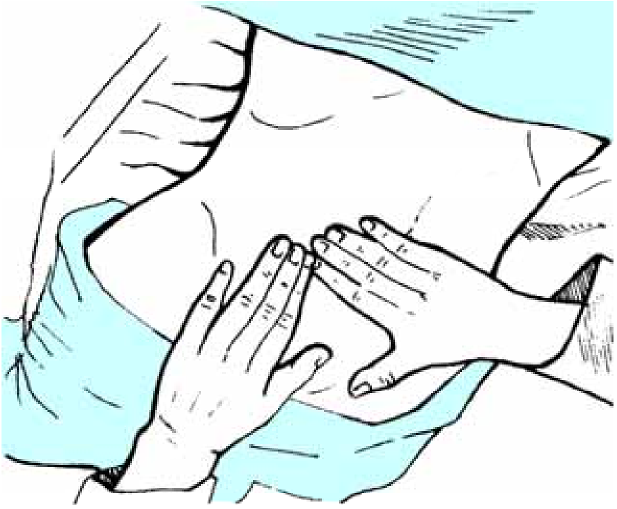
Devotee Palpation

Same as the previous one, but with the patient standing, in order to provide descent of the organ and make the method more sensitive (fig. 61.10).

Glenard bimanual palpation (thumb palpation)

It consists of placing the left hand hugging the lumbar region with the four fingers behind and the thumb in front, which falls below the costal margin. The right hand on the anterior wall allows exploration of the inferior border (fig. 61.11).

Fig. 61.8 Gilbert's bimanual palpation.



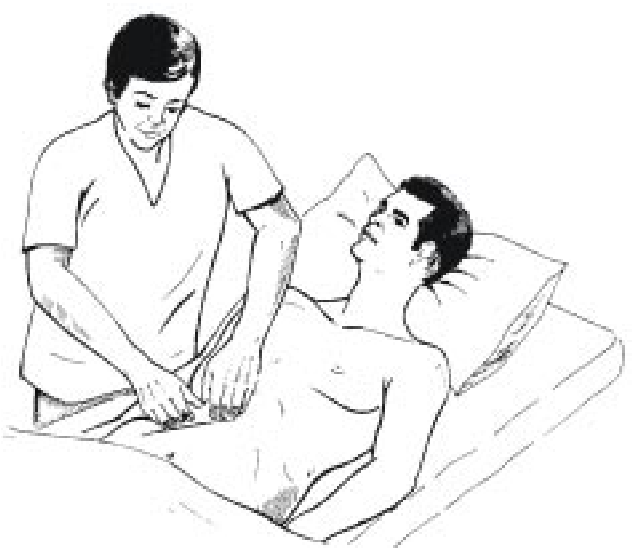


Fig. 61.9 Mathieu's bimanual palpation.

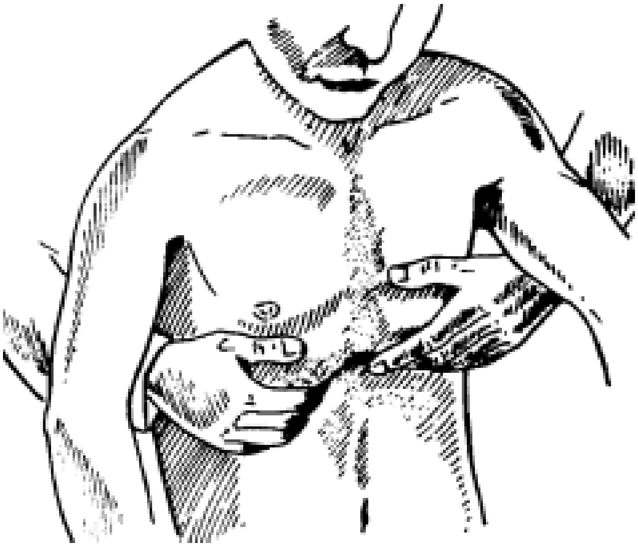


Fig. 61.10 Bimanual palpation of Devoto.

Palpation of the gallbladder

There are also different maneuvers that aim to bring the gallbladder closer to the palpating hand and cause pain:

- Murphy maneuver.
- Abraham's maneuver.
- Fiessinger maneuver.

Murphy maneuver

It consists of inserting the fingers of the right hand like a hook under the costal margin at the level of the gallbladder. Other authors, such as Pron, preferred to use both thumbs at the level of the vesicular point and invite the patient to inhale (fig. 61.12).

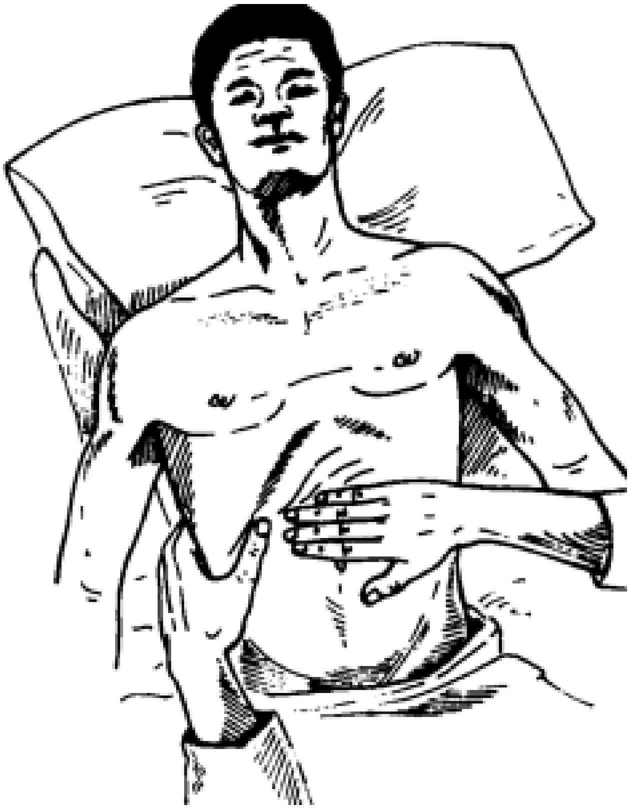


Fig. 61.11 Glenard bimanual palpation.

Abraham's maneuver

To perform this author's maneuver, the patient is placed in a supine position, the midpoint of the line that goes from the ninth cartilage to the navel is sought, and by sinking one or two fingers of the right hand here, severe pain is caused in patients with calculous vesicles.

Fiessinger maneuver

The entire hand is applied to the right hypochondrium and the patient is invited to breathe deeply and gently, thus causing stabbing pain in calculous cholecystitis.

Palpation of the pancreas

In general, it is considered very difficult due to the deep location of the organ; However, we use three methods:

Grott method.

- Mallet-Guy method.

Desjardins pancreatic point.

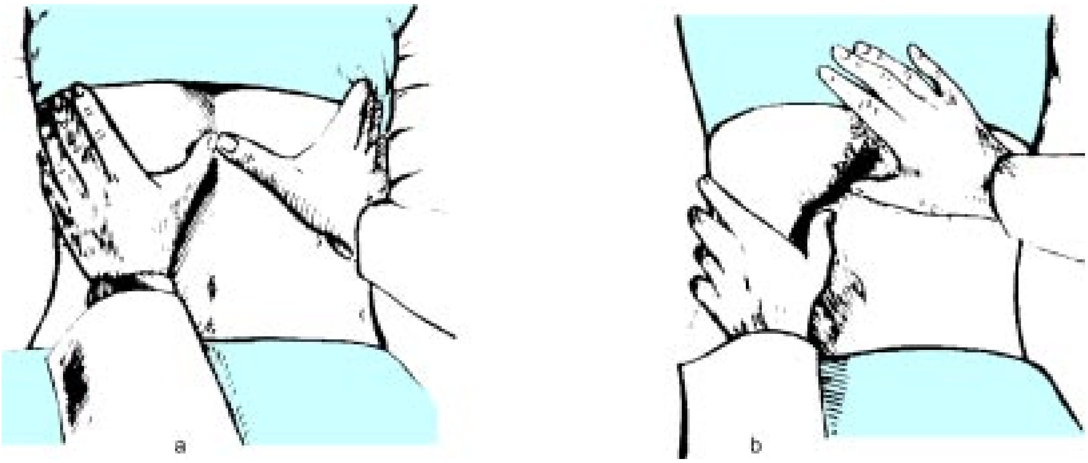


Fig. 61.12 Pron maneuver: a, with both thumbs placed below the costal arches, gentle pressure is applied to them in order to examine local sensitivity to it; b, the provocation of pain when taking a rapid and deep inhalation, at the same time as pressing on the gallbladder area, speaks in favor of the involvement of the cholecyst or bile ducts (Murphy's sign), if the character of the former corresponds to the spontaneous discomfort of the patient.

Grott's method

The patient is placed in supine position with legs flexed. A small roller or pillow is placed under the lumbar spine to cause lordosis, which brings the pancreas closer to the anterior wall and relaxes the muscles. The right hand of the explorer, placed on the external edge of the rectus femoris, pushes it towards the midline, allowing the pancreas to be explored in depth at the level of its junction with the abdominal aorta and the vertebral column (fig. 61.13).

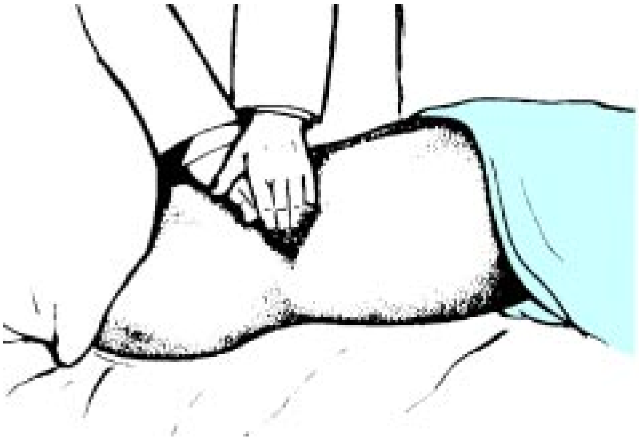
Mallet-Guy method

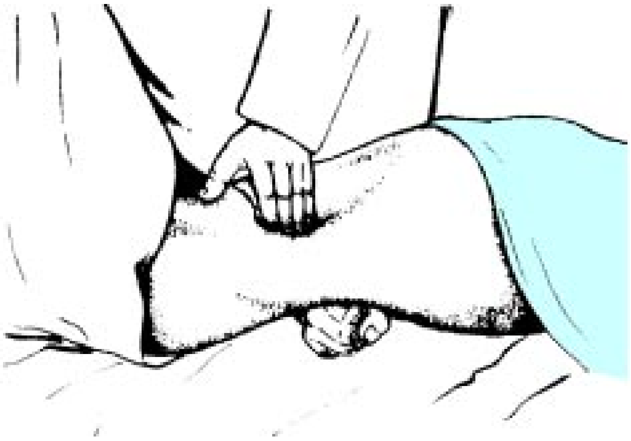
To palpate the body and tail of the pancreas, this method is recommended, in which the patient is placed in the right lateral decubitus position with the thighs semi-flexed over the abdomen. The doctor places his right hand so that is located in front of the ninth cartilage, at an approximate distance of 3-4 cm from the costal margin, the anterior abdominal wall is depressed by placing the fingers under the costal grille, in the direction of the left laterovertebral region. Finally, the base of the hand is raised and the fingers are sunk deep above the stomach, which is rejected to the right. The purpose is to circumvent the obstacle represented by the stomach and thus cause deep pain in the pancreas (fig. 61.14).

Desjardins pancreatic point

It is described 6 cm from the navel on a line that joins it with the right armpit; corresponds to the Wirsung duct at its duodenal outlet (Fig. 61.15).

Fig. 61.13 Palpation of the pancreas according to Grott.





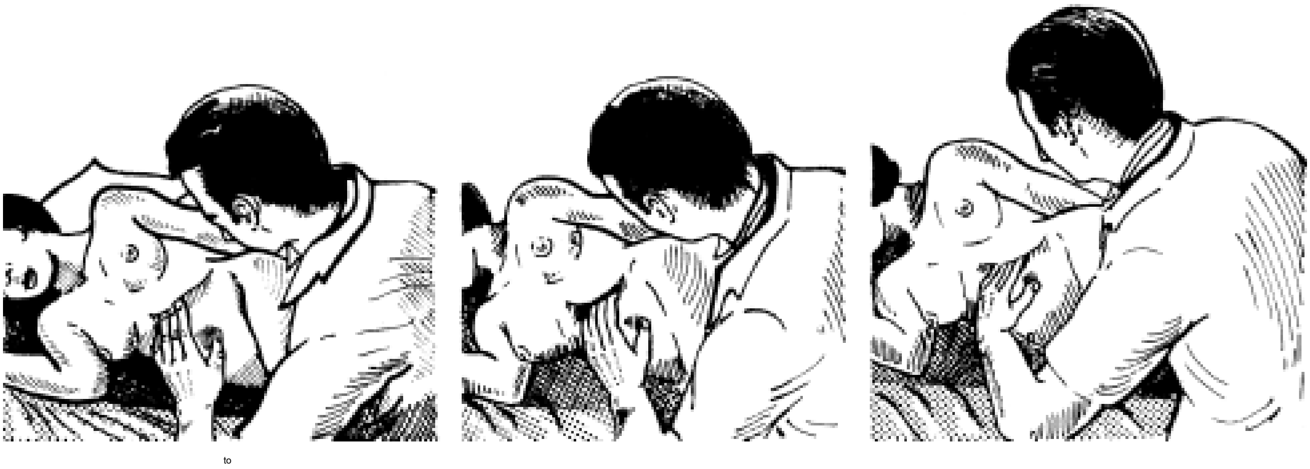


Fig. 61.14 Investigation of the pancreatic tender point of the left upper quadrant or Mallet-Guy method: a, the subject is placed in the right lateral decubitus position with the thighs flexed; b, with the end of the observer's fingers located 3 or 4 cm from the costal margin, it is placed parallel to the superficial plane, under the rib cage; c, then, turning towards the depth, palpate the pancreas directly, above the stomach reclining to the right.

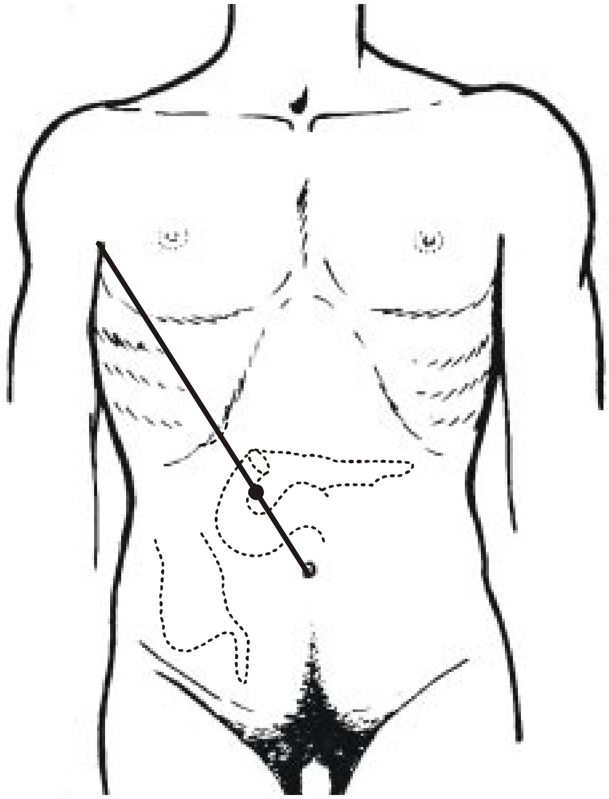


Fig. 61.15 Desjardins pancreatic point.

Liver percussion

It is carried out using Gerhardt's digital-digital method, delimiting two areas: upper and lower (which we will describe in the following section). It is struck in the anterior or lateral plane. In the pulmonary interposition zone, the percussion must be strong, deep; in the free zone, it must be superficial.

NORMAL DATA

When palpation is performed in normal subjects with little fat, it is possible to identify the lower border of the liver. Through percussion, an upper zone of relative dullness is identified, which corresponds to the hepatic portion covered by the rib cage, which is related above to the pleuropulmonary cul-de-sac, and a zone of absolute dullness, which corresponds to the lower portion of the liver in contact with the abdominal wall, particularly when descending during inspiration.

Up to the IV or V ribs, lung sonority is obtained, and from that level onwards, first relative dullness is achieved, then absolute dullness (fig. 61.16).

Normally, data are not obtained from the bile ducts or the pancreas.

PATHOLOGICAL DATA

Due to its importance, we will study:

- Spasticity or parietal contracture.
- Hepatoptosis or descent of the liver.
- Hepatomegaly.
- Caused pain.
- Vesiculomegaly or large palpable vesicles.
- Increase in volume of the pancreas.
- Vascular beats.

Spasticity or parietal contracture

We must point it out as the first fact, once again, since it indicates the presence of an acute process at the level

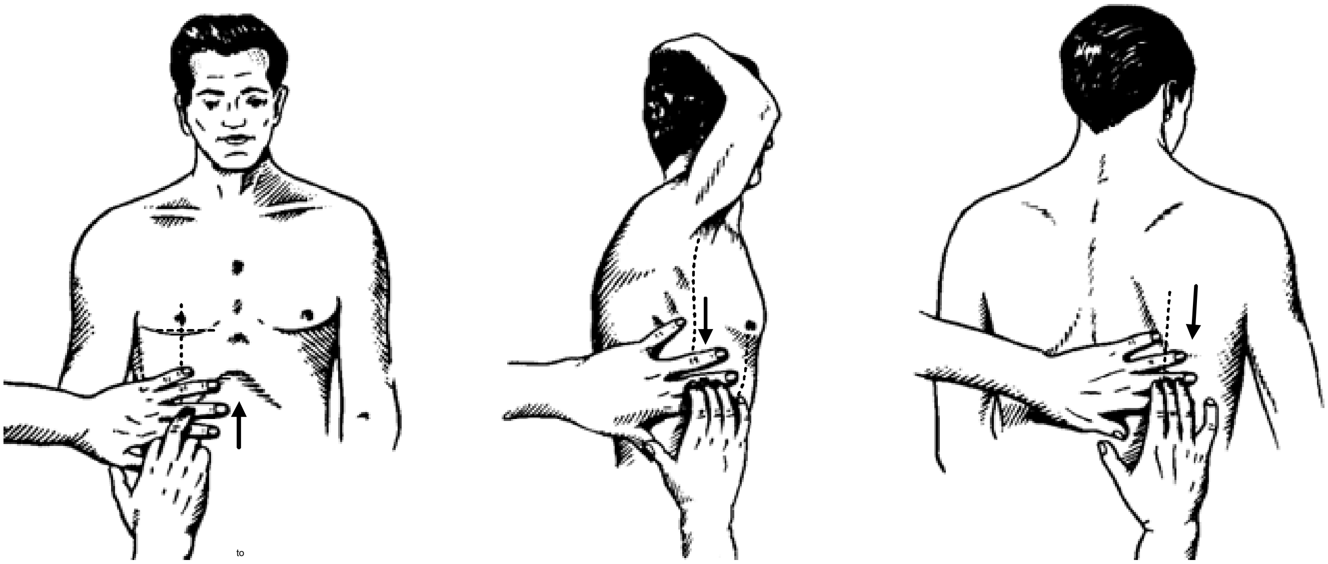


Fig. 61.16 Percussion of the liver in the planes: a, anterior; b, side; c, posterior.

of the right hypochondrium. Extension towards the epigastrium is common, especially towards its right half. It indicates, as we have repeated several times, that there is a peritoneal inflammatory reaction.

It occurs in acute, viral or parasitic hepatitis and liver abscesses. It can also be caused by acute cholecystitis, generally lithiasis.

It is possible to verify skin hyperesthesia, as well as the presence of a limited, cake-shaped plastron or thickening, which is seen when the acute process encompasses the loops and omentum.

Hepatoptosis or descent of the liver

In this case, the liver is palpable below the costal margin, but maintains its normal characteristics. In turn, it is easily distinguished from hepatomegaly by the downward displacement of the upper edge, as we will see in the pathological percussion.

Hepatomegaly

They constitute a true syndrome caused by multiple causes. They are observed during inspection, when they are very marked, and are more easily identified on palpation, which allows us to orient ourselves to their possible cause, by being able to study their characteristics: consistency, size, sensitivity. mobility, regularity, form, etc. We do not intend to make a complete review of hepatomegaly, we will only list the most important ones in semiological order and their palpatory characteristics:

a) Benign and malignant tumors. Malignant ones are more common, including secondary or metastatic ones. In the latter, hepatomegaly is large, hard, woody and generally nodular.

b) You heard. With a smooth surface when they are single or multinodular when there are many, they always present the sensation of reluctance if they are carefully palpated (fig. 61.17).

c) Cardiac liver (passive congestion). Smooth, painful and variable in size, parallel to heart failure (accordion liver).

d) Leukemic liver. Smooth, sometimes large, particularly in myeloid leukemia.

e) Cirrhotic liver. It can adopt various modalities during palpation, but there are some common characteristics such as: increased consistency, which is firm or hard, and a thin, sharp edge, which jumps upon contact with the hand.

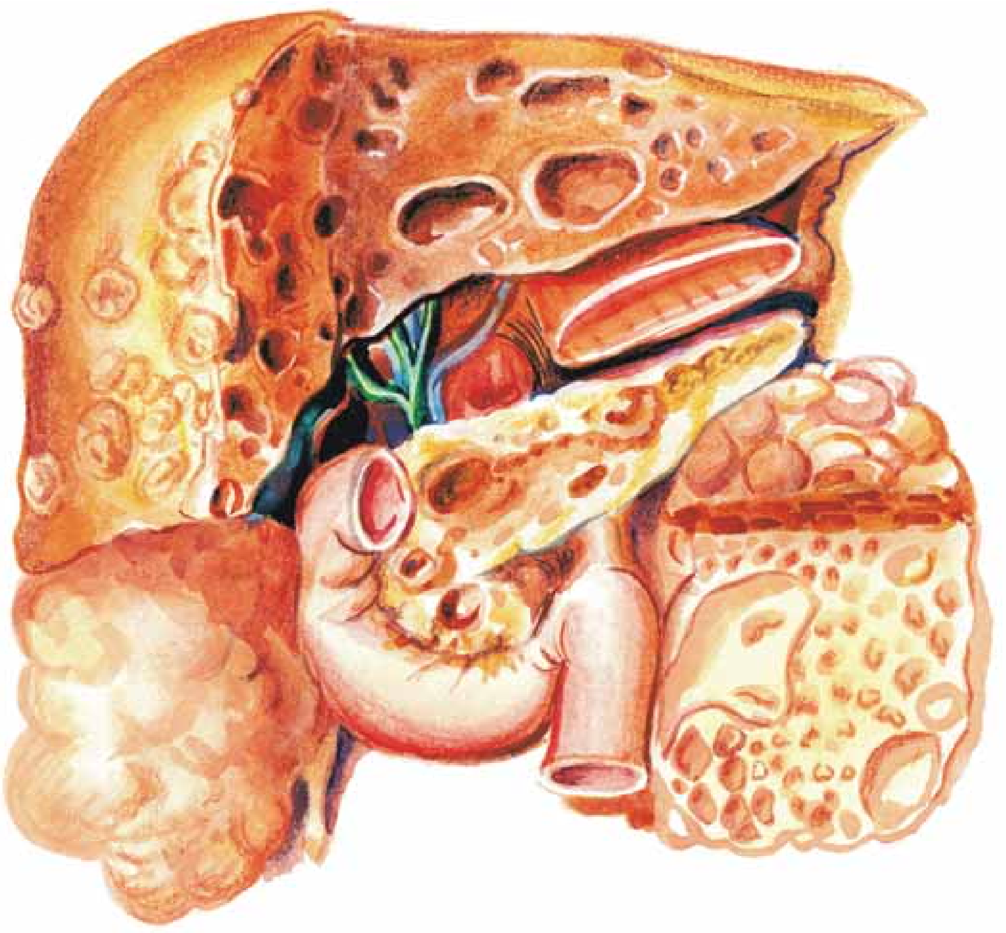
According to the type of cirrhosis and its evolutionary stage, we will point out some particular characteristics:

Cirrhosis in the hypertrophic phase (Fig. 61.18) (post-necrotic, portal or biliary) in which the liver is enlarged, with a smooth surface on palpation, except in subjects with a very thin wall in whom it is possible to palpate an irregular surface; However, it is worth noting that small accumulations of fat in the wall are often taken as nodules, which leads to erroneous diagnoses (an aspect that Llanio believes should be insisted upon to avoid mistakes).

Cirrhosis in the atrophic phase presents a harder, smaller liver with a very thin and sharp edge (fig. 61.19).

Macronodular post-necrotic cirrhosis presents medium-sized (1-3 cm in diameter) and large nodules, which can be confused with neoplasms (Fig. 61.20).

Fig. 61.17 Liver
polycystic



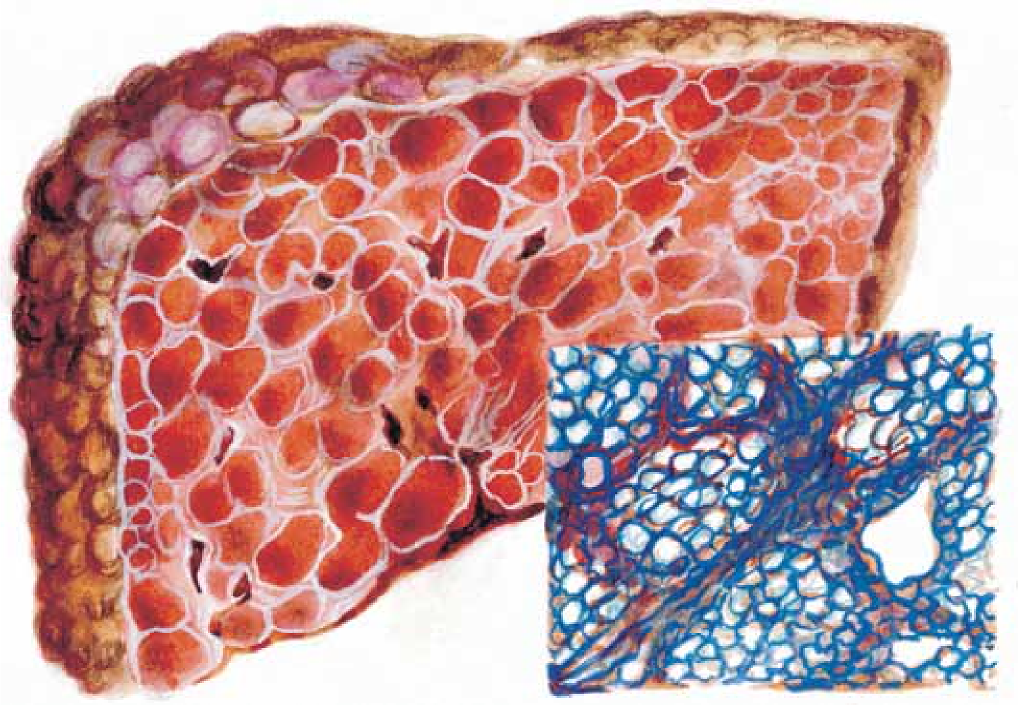
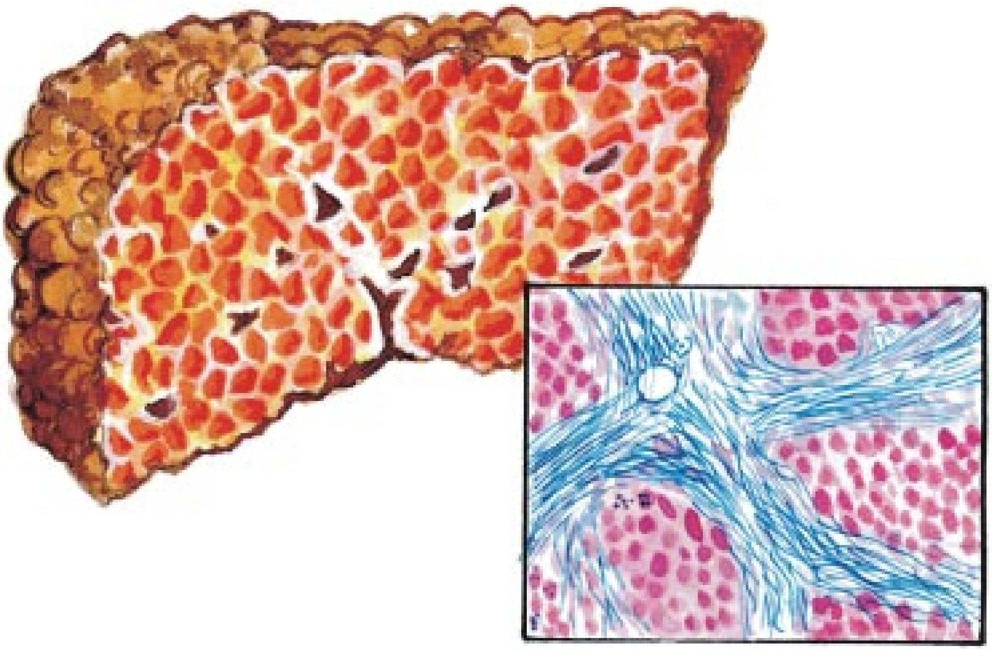


Fig. 61.18 Cirrhosis in
hypertrophic

Fig. 61.19 Cirrhosis in atrophic phase.



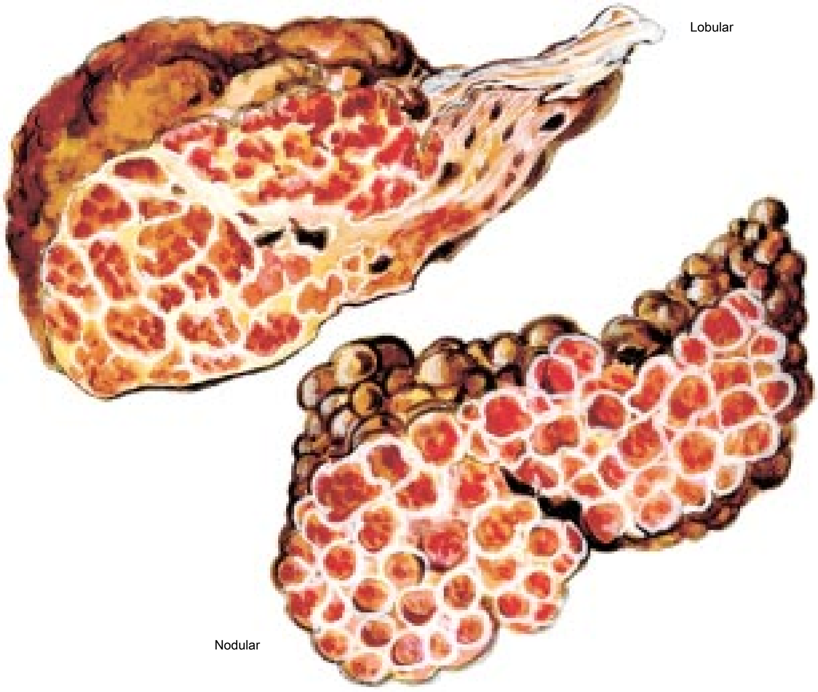
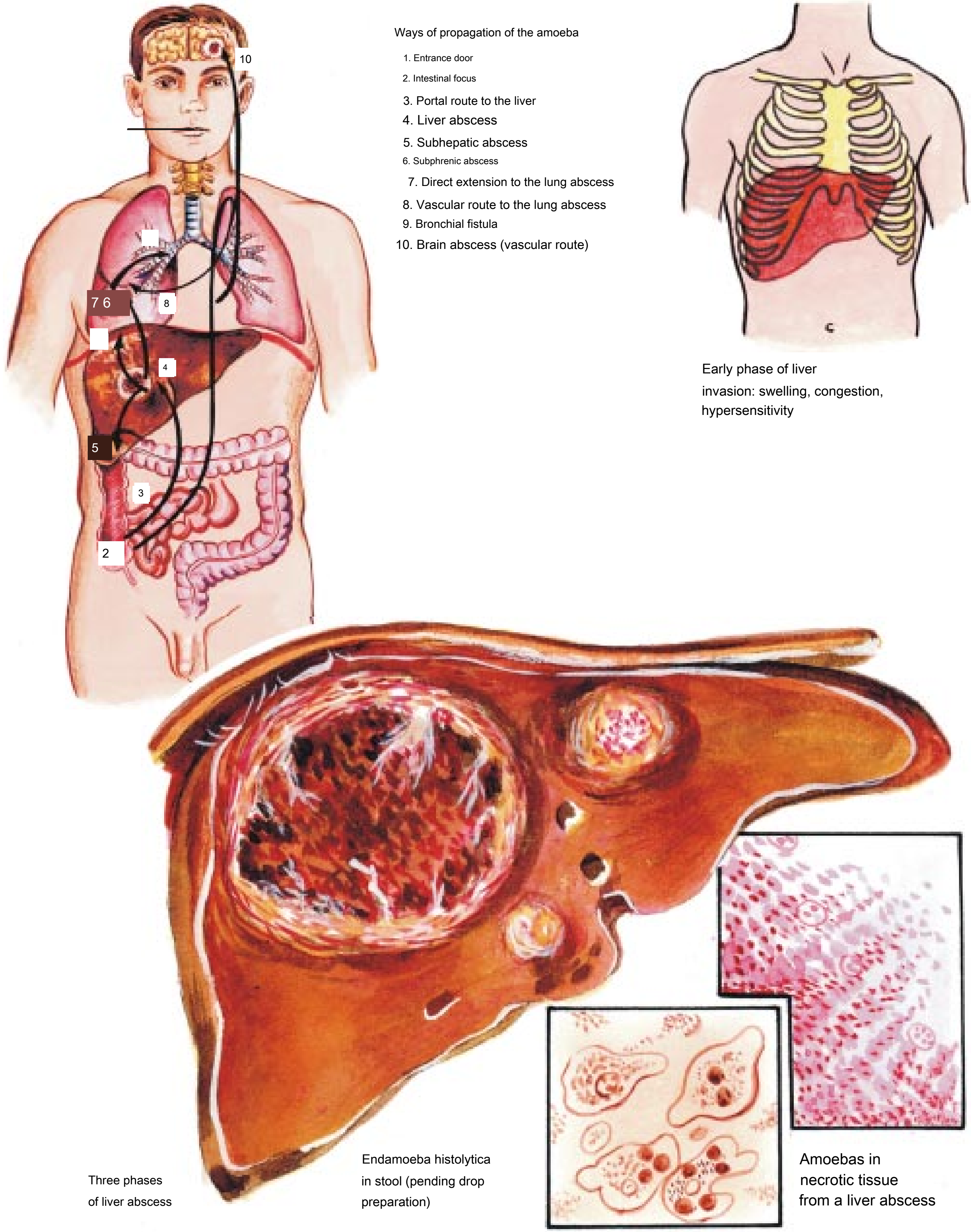


Fig. 61.20 Macronodular cirrhosis.



Ways of propagation of the amoeba

1. Entrance door
2. Intestinal focus
3. Portal route to the liver
4. Liver abscess
5. Subhepatic abscess
6. Subphrenic abscess
7. Direct extension to the lung abscess
8. Vascular route to the lung abscess
9. Bronchial fistula
10. Brain abscess (vascular route)

Early phase of liver invasion: swelling, congestion, hypersensitivity

Three phases of liver abscess

Endamoeba histolytica in stool (pending drop preparation)

Amoebas in necrotic tissue from a liver abscess

Fig. 61.21 Parasitic hepatomegaly. Abscess.

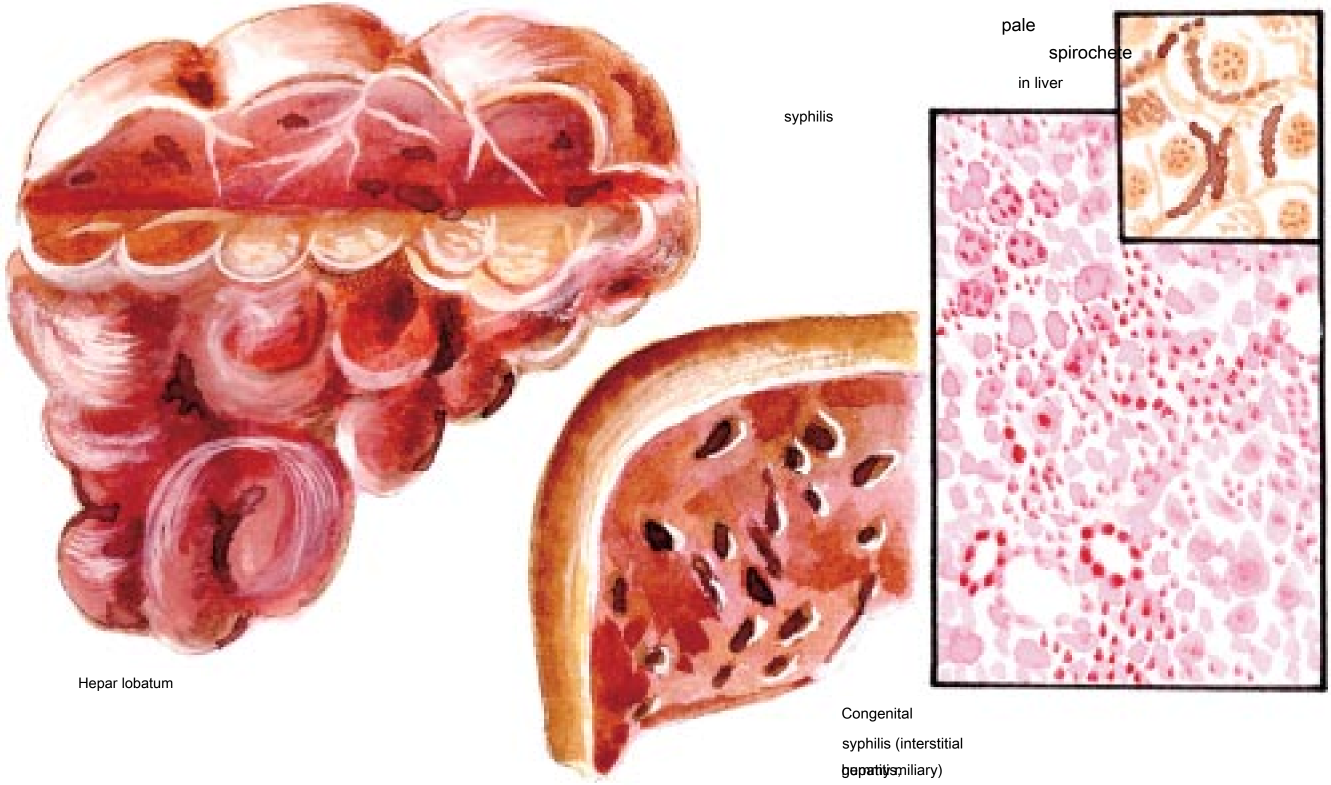


Fig. 61.22 Syphilitic hepatomegaly.

Let us also point out that in this type of cirrhosis sometimes the lesion predominates in one lobe. In many cases the other is palpated greatly enlarged, in a vicariant function.

f) Inflammatory liver (active congestion). It is smooth and painful, typical of acute hepatitis (viral, for example): however, hepatomegaly is discreet.

g) Parasitic hepatomegaly (amoeba, fasciola, etc.). The liver is large, smooth, very painful, sometimes fluctuating if there is an abscess accessible to the hand (fig. 61.21).

h) Syphilitic hepatomegaly. More rare, irregular, hard, with a "stringed" liver (ficele), or thick nodular (heparlobatum) (fig. 61.22).

i) Other hepatomegalies. These are those found in: amyloidosis, malaria, hemochromatosis, fatty liver, cholostatic liver due to biliary obstruction and thesaurismoses. In all of them the liver appears voluminous, smooth, firm and with a blunt edge.

Fortunately, the semiological interpretation of hepatomegaly is facilitated when the associated symptoms are assessed: splenomegaly, ascites, jaundice, etc., which corresponds to the syndromological study.

Caused pain

When limited to the right hypochondrium, it is the expression of the Morley reflex and indicates the externalization or serous reaction of processes corresponding to this area. It is common in acute hepatitis with perihepatitis, for example, amoebic and sometimes viral type, and in cardiac liver, among others.

Pain caused by acute cholecystitis, whether lithiasis or not, is seen. The painful points are multiple: cystic, right epigastric; and outside the region we must point out the points: phrenic, supraclavicular, deltoid and mammary, scapuloapexian and, in the spinal column, from D8 to Dll.

Vesiculomegaly or large palpable vesicles

They may correspond to Courvoisier's mechanism or law, as observed in cases of obstructions of the common bile duct or ampulla of Vater (generally due to neoplasms) and whenever there is a healthy gallbladder. It is known as the Courvoisier-Terrier sign and is associated with cholestatic hepatomegaly (that is, with bile stasis) (Fig. 61.23). The vesicle retains its shape (piriform), mobility, resistance, etc., making itself accessible in the hypochondrium and even descending to the right flank or void. We must also point out the pyocholecyst or empyema of the gallbladder and the hydrocholecyst, with its stringy, mucoid content (white bile), of occlusions of the cystic duct; and some malignant tumors of the gallbladder (in this case, the palpable tumor is hard).

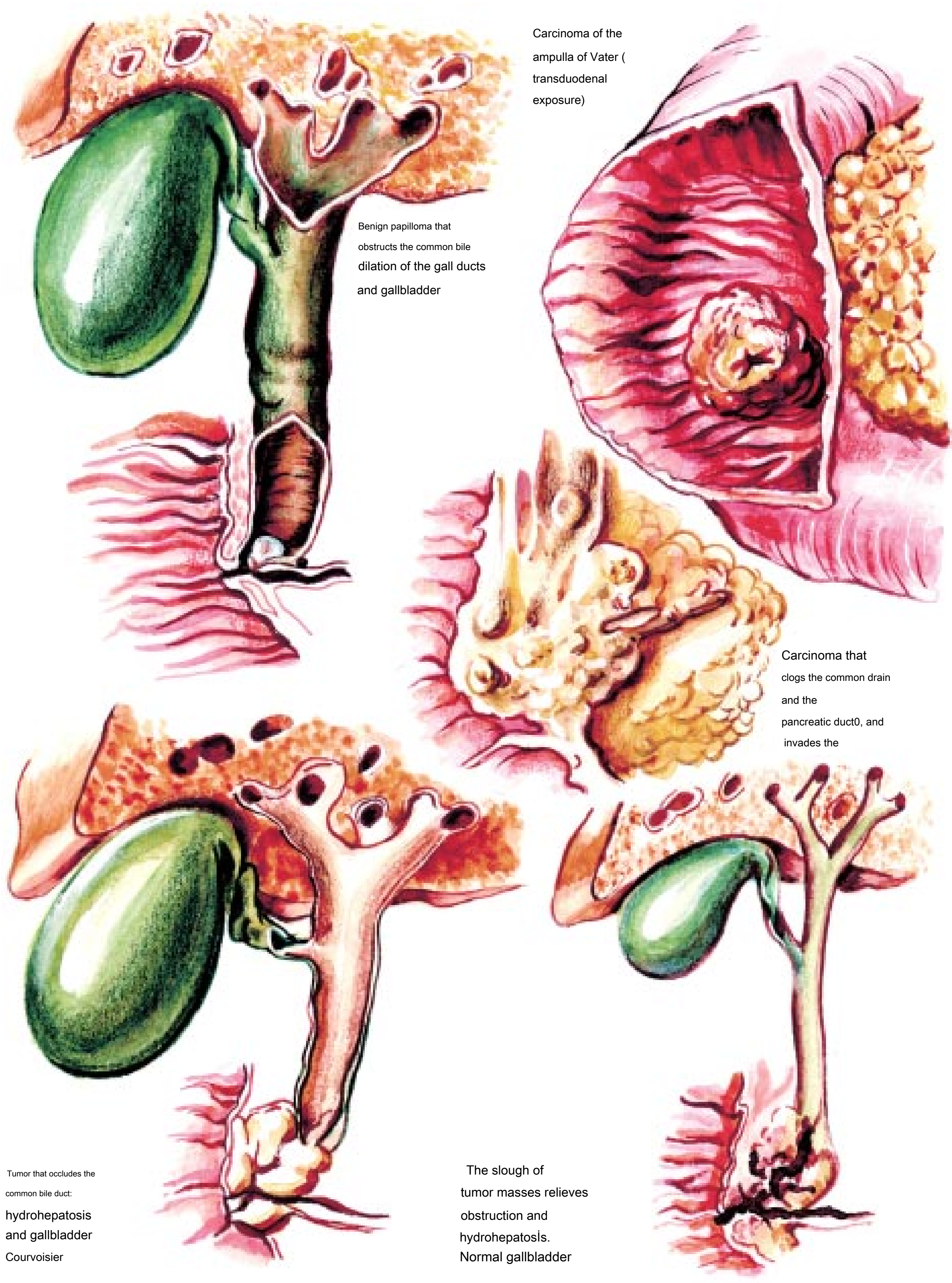

Carcinoma of the
ampulla of Vater (
transduodenal
exposure)
Benign papilloma that
obstructs the common bile
dilation of the gall ducts
and gallbladder
Carcinoma that
clogs the common drain
and the
pancreatic duct0, and
invades the
Tumor that occludes the
common bile duct:
hydrohepatosis
and gallbladder
Courvoisier
The slough of
tumor masses relieves
obstruction and
hydrohepatosİs.
Normal gallbladder

Fig. 61.23 Vesiculomegaly.

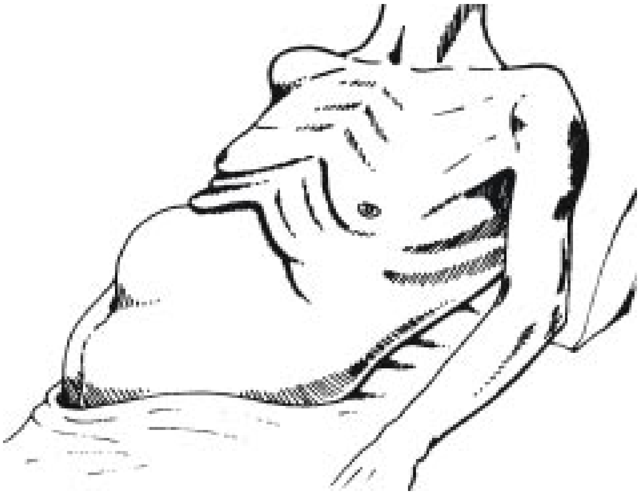


Fig. 61.24 Pancreatic cyst displacing the stomach upwards and the transverse colon downwards.

Increased volume of the pancreas

Only in those cases where a pathological pancreas makes contact with the anterior wall, can physical or objective symptoms be obtained.

In these circumstances we can find:

On inspection, vaulting of the epigastric region (in the center or on the right) produced by tumors or cysts that sit in the head of the organ.

They are sometimes seen at lower levels, especially in very large cysts in the body or tail of this organ (Fig. 61.24).

On palpation, in similar circumstances, the firm or hard sensation is appreciated in tumors and the characteristic retentive sensation in cysts that do not move due to changes in position or manual maneuvers. It is possible to find, on deep palpation, variations in consistency in some cases of chronic pancreatitis, as well as greater ease in the transmission of aortic beats to the palpating hand (Körte sign). Finally, the pain caused by the pancreas is described, both in especially acute pancreatitis and in tumors. The pain corresponds to the points and areas described previously (Desjardins pancreatic point and Chauffard's pancreaticocholedochal zone), as well as to Katsch's hyperalgesic zone, in the left hemibelt, at the level of D10 to D12 in acute pancreatitis. Mallet-Guy has advocated a method of palpation in which he tries to overcome the obstacle represented by the stomach, already described (see fig. 61l.14).

Percussion and auscultation do not provide data of interest.

To finish the particular study of the right hypochondrium, let us mention the possibility of finding physical symptoms corresponding to pathological processes of the angle. right of the colon (colitis and tumors) and its immediate portions: highest portion of the ascending colon and rightmost portion of the transverse colon.

Finally, we must remember the possibility of tumor processes of the right kidney and adrenal gland, although these organs do not belong to the digestive system.

Vascular beats

They correspond to the so-called hepatic pulse; between them have:

a) The positive, true heartbeat (systolic), caused by blood reflux that expands the liver, typical of tricuspid insufficiency.

b) Hepatic transmission of the aortic heartbeat.

c) The negative beat that is sometimes observed in the epigastrium (Brums' epigastric beat), typical of right ventricular hypertrophies.

In percussion the pathological data are more limited. As we said in previous paragraphs, we diagnose hepatic ptosis by checking the descent of the upper limit of the liver, simultaneously with palpation of the viscus below the rim. In cases of large hepatomegaly, it is possible to percuss the upper edge above its normal limit. This percussive limit of the liver can be altered by extrinsic pathological processes or by the organs of the thorax: lung, pleura, etc.

The hepatic percussive zone is reduced or disappears in cases of acute or chronic liver atrophy, which has a high diagnostic value. In cases of perforation of an abdominal viscus, especially due to an ulcer, the dull area on liver percussion disappears. The same occurs in pneumoperitoneum. Finally, the descent of the lower border, although percussable, is generally better determined by palpation. This decrease occurs in hepatomegaly; when the liver is pushed up and down by large effusions; in subphrenic abscesses and in tumors of the pleural cul-de-sac.

On auscultation, exceptionally, rubs corresponding to perihepatitis, or murmurs due to tricuspid lesions or liver angiomas, can be perceived.

The gallbladder and the rest of the biliary system do not provide pathological data on percussion and auscultation.

EXAMINATION OF THE TRANSVERSE COLON, THE JEJUNUM, THE ILEUM AND THE MESOGASTRIUM

CLINICAL ANATOMY

The transverse colon projects slightly above the umbilicus following a somewhat horizontal line.

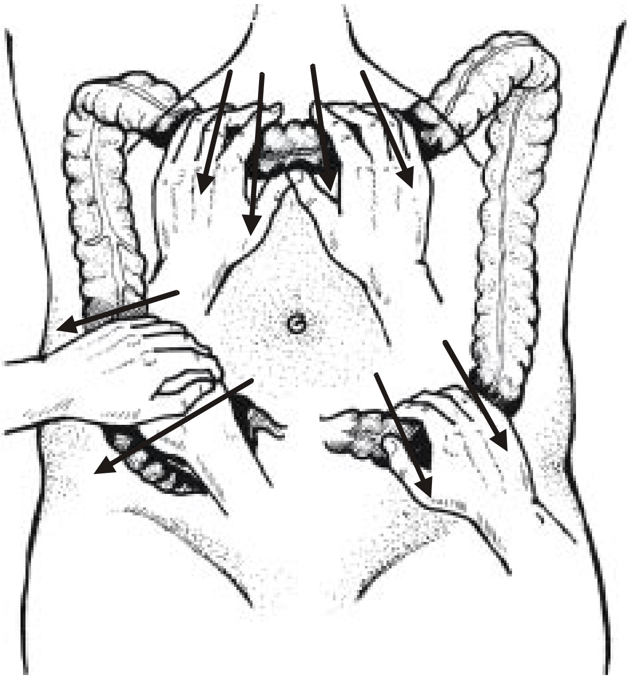
concave upward, extending from right to left and ascending at its left end to the level of the last costal cartilages. Let us remember, however, the great mobility, width and laxity of the support means of the transverse colon, which explains why we can find it above and below the indicated region.

The central mass of the small intestine generally includes intermediate and final loops of the jejunum, as well as initial and intermediate loops of the ileum whose intestinal mass projects primarily into the mesogastric area that we are studying. The initial loops of the jejunum commonly project into the left hypochondrium, and the terminal loops of the lion into the right void and iliac fossa on the same side. In any case, let us remember the great mobility of the jejunum and ileum and therefore the possibility that one of its handles can be projected anatomically in any abdominal area.

NORMAL DATA

Palpation of the transverse colon is your best method of exploration. It is done by the bimanual method and by sliding, taking advantage of the spinal column as a plane of resistance. The tracing is carried out from the epigastrium to the mesogastrium, reaching the transverse colon in the midline and, with the last three fingers of each hand, we try to reach its right and left lateral ends. It is identified as a cylinder (fig. 61.25).

Fig. 61.25 Correct technique for palpation of the transverse colon, cecum and sigmoid loop.



Palpation of the jejunum and ileum is performed by the monomanual or bimanual method, but in most normal cases the loops are not palpated.

Percussion and auscultation vary in relation to the digestive period and sometimes to the type of food ingested. Through percussion we can find discrete degrees of tympany and subdullness, and through auscultation hydroaerial noises characteristic of the peristalsis of the jejunum and phleon during the intestinal phase of digestion. It is advisable to become familiar with the characters of these noises, as well as their frequency.

PATHOLOGICAL DATA

On inspection, only vaulting due to large tumors and cysts, especially of the mesentery, are important. Also of interest are visible peristaltic movements of small bowel obstructions.

On palpation, the transverse colon appears reduced in caliber, hard and painful like a rope in organic colitis and functional colopathy (irritable colon); Instead, it provides a sensation of increase with an irregular and sensitive surface in processes of a tumor nature, whether inflammatory or neoplastic.

The small intestine can also be the seat of tumors, as well as the greater omentum and the root of the mesentery. These tumors are generally mobile vertically or transversely; Sometimes an elongated hard formation in the shape of a black pudding is palpated in cases of intestinal intussusception, which, together with tympanism and increased fluid sounds, allows the diagnosis of this entity to be made. We also consider umbilical pathology in the mesogastric area, already mentioned for the general physical examination of the abdomen. It is possible to verify parietal contracture in acute processes.

The pain caused is obtained in transverse segmental colitis, in tumors and in other inflammatory processes; It may also correspond to the small intestine and the mesos. Different painful points of the small intestine have been mentioned that we do not transcribe due to their inaccuracy. The signs obtained in percussion by Mathieu and in auscultation by Wilms corresponding to dilations of the small intestine are equally fallacious.

However, sometimes a differential diagnosis is made between mechanical ileus and paralytic ileus through auscultation, as we noted in the previous chapter.

In the first, the presence of frequent and intense air-fluid sounds is confirmed, in contrast to their absence in paralytic ileus.

Let us remember that in the differential diagnosis, the physical alterations obtained in the deep portion of the mesogastrium must be taken into account, corresponding to the great vessels, ganglia and nervous plexuses of the region.

EXAMINATION OF THE ASCENDING COLON AND VOID 0 RIGHT FLANK

CLINICAL ANATOMY

The ascending colon projects mainly into the void or right flank, extending towards the iliac fossa and hypochondrium on the same side.

NORMAL DATA

Palpation can be monomanual or bimanual and by sliding; The rally maneuver can also be performed in that area to distinguish renal palpation; Identification of the normal ascending colon is not easy. Percussion in its normal state gives a tymponic sound.

PATHOLOGICAL DATA

Let's point out in the inspection, the large tumors. Benign tumors, malignant tumors, and inflammatory infiltrative processes are identified on palpation. Kidney tumors are also projected here.

The pain caused corresponds to colitis of the region, tumors and other inflammatory processes. The skilled hand can notice spastic areas limited to a certain level of the ascending colon.

On percussion, a large meteorism can be identified, which corresponds to a dilation, due to partial obstruction according to Von Wahl's law, or due to megacolon.

Finally, we must take into account that in the void area or right flank, physical symptoms corresponding to similar processes of the small intestine, especially the ileum, can also be seen.

EXAMINATION OF THE DESCENDING COLON AND VOID LEFT FLANK

CLINICAL ANATOMY

The descending colon projects from the left hypochondrium, occupying the void, to the left iliac fossa.

NORMAL DATA

Palpation is done exactly the same as for the other flank. The lower third of the normal descending colon is often palpable.

PATHOLOGICAL DATA

We can apply everything stated in parallel to the right void, but of course referring to the descending colon. Let us mention in particular, chorda colic and fecalomas; The sign of the godet is very important These leave pressure which allows them to be differentiated from other tumors. Although the frequency varies in different diseases, in one portion of the colon or another the pathology is similar. Although the sigmoid loop of the colon partially projects into the lower portion of this area, we will study it in the hypogastrium.

Finally, we must take into account the ileum and jejunum in this area.

EXAMINATION OF THE SIGMOID LOOP OF THE COLON AND HYPOGASTRIUM

CLINICAL ANATOMY

The sigmoid loop projects into the lowest portion of the left void, as well as, fundamentally, into the hypogastric area.

NORMAL DATA

Palpation can be monomanual or bimanual and can be completed by performing a rectal or vaginal examination at the same time, which greatly facilitates exploration. It is difficult to palpate this segment of the colon in a normal state.

PATHOLOGICAL DATA

Although some authors refer sigmoid pathology to neighboring superior regions, it can also be identified in the hypogastrium. Let us point out tumors, occlusions caused by volvulus or rotation of the sigmoid on its axis, pericolitis, pain caused by sigmoiditis, etc.

In the hypogastric zone, it is important to take into account, in the differential diagnosis, female bladder and genital pathology, which has been studied in the semiology of these systems.

EXAMINATION OF THE LOWEST PORTION OF THE DESCENDING COLON, THE SIGIMOID LOOP AND THE LEFT ILIAC FOSSA

CLINICAL ANATOMY

Let us mention the projection of the lowest or terminal portion of the descending colon, as well as the projection of the highest portion of the sigmoid loop of the colon and the colonic segment already studied in the hypogastric area, where the lowest portion of said sigmoid loop is projected.

NORMAL DATA

Palpation is done by the monomanual or bimanual method using only three fingers, which is favored by the contracture of the psoas muscle and is completed by rectal and vaginal examinations, if necessary.

The upper portion of the sigmoid and the lower portion of the descending can be palpated in the form of a more or less firm cylinder in normal cases.

PATHOLOGICAL DATA

On inspection, less frequently than in the right iliac fossa, vaulting due to large tumors can be seen, particularly of the descending and sigmoid or its meso.

On palpation, intense contracture of the area is confirmed in acute sigmoiditis and in diverticulitis with peritoneal reaction. Let's mention the tumors of that segment of the colon (benign or malignant). Perisigmoiditis can also present a palpable tumor, as can fecalomas and sigmoid volvulus. There is pain caused in the region by inflammatory processes, verifying the well-known Lane sign of the colic chord in multiple processes accompanied by contracture of this colonic segment, both functional and organic.

In this area, we must also take into account the alterations of the genitals in women, the ureter, the psoas and the plexuses and vessels.

EXAMINATION OF THE CEUM, THE APPENDIX AND THE RIGHT ILIAC FOSSA

CLINICAL ANATOMY

The cecum projects into the right iliac fossa with the variation corresponding to the constitutional type of the patient, in which it will be located higher or lower.

The appendix, an organ of great importance due to the relative frequency of its acute inflammation in daily clinical practice, projects predominantly from the cecum, oriented downwards, but we must take into account its variants of direction: inwards, upwards, downwards, outwards and especially behind the cecum itself (the so-called retrocecal appendix), whose anatomical variants have great semiological importance. We will point out later the areas and points of particular projection.

NORMAL DATA

Palpation of the right iliac fossa, particularly referring to the cecum and appendix, is very rich in maneuvers. bras. It is performed by monomanual or bimanual methods.

The cecum is palpated by tracing, from the navel towards the anterior superior iliac spine (from inside to outside) and vice versa, preferably with the lower limbs extended. The patient can also be placed in the left lateral decubitus position and then the right lower limb is made to flex. You can use one or both hands. Sigaud advises using the left hand with the thumb behind and the other fingers in front. The clawed fingers, with slight flexion, perform the sliding described by Haussmann and Glenard, detailing the cecum (see fig. 61.25).

The normal cecum is recognized by the following characteristics: shape and size like that of a pear, with the part widened downwards, soft consistency, smooth surface, movable 2-4 cm inwards, painless, and when palpated it gives rise to the production of hydro-aerial sounds Ogurgoteo, due to the liquid-gas collision of the contents.

These characteristics vary according to the state of contraction or relaxation of its muscular tunic and according to the amount of liquids or gases contained inside; Precisely this variability is a very important element for its identification and differentiates it from a ptotic kidney or a large gallbladder.

The terminal ileum can sometimes be palpated as a thin rope.

In the area itself, the technique of palpation of the psoas muscle is important: with the patient in the supine position, the contraction of the muscle is sought at the expense of the lifting and flexion movement of the limb.

Vaginal and rectal examination are very useful to complete the best of palpations, since the abdominopelvic fornices are explored and, with experience, alterations due to the cecum and appendix are distinguished from those due to the genital and urinary organs.

Palpation of the appendix itself is impossible, even though the abdominal wall is thin and hypotonic.

In summary, it is normally possible, in some subjects, to palpate the cecum and even the terminal ileum, but not the appendix.

PATHOLOGICAL DATA

Muscle contracture is common in this region due to the acute processes that occur there. It is then difficult for the doctor to obtain details, which is why in these cases he uses vaginal and rectal examinations to guide the diagnosis. Of course, the mere fact of the intense contracture (acute right iliac fossa) forces us to think about inflammatory processes with a peritoneal reaction, among which we preferably have acute appendicitis.

Let us also point out typhlitis, adnexitis, tubal pregnancy, psoitis, acute ileitis, among others.

Sometimes, acute appendicitis not diagnosed in time leads to the formation of a plastron (septum perforation), which is palpated as a hard, irregular and painful tumor mass.

Let us now refer to the pain caused in the right iliac fossa, which is observed in the areas and points that we describe below and which are due to variations in the position of the appendix and the cecum.

Saint'Angelo delimits the appendicular area in the following way: inside, by a line that goes from the navel to the pubic symphysis; below and outside, by a line that goes from the crural arch and the iliac crest to the external edge of the square of the loins: above, by the horizontal line that passes through the navel and that also reaches the aforementioned muscle behind. As can be seen, this area is narrow at the back and widens as it approaches the midline. All appendicular points would be included within this area (fig. 61. 26).

On the line that goes from the navel to the anterior superior iliac spine are the Morris points 3-4 cm from the navel; that of Monro, at the intersection of said line with the anterior rectum, and that of Mc. Burney in the middle of said line. On the line that joins both iliac spines, there will be the Lanz point, at the junction of the right third with the middle and the Sonnenburg point at the intersection of said line with the rectus femoris. The most frequent painful points are those of Lanz, Mc. Burney and Morris. There are other maneuvers that cause pain, such as: Rowsing's, which consists of applying pressure from the left iliac fossa, following the direction of the colon in the opposite direction. The idea is to cause distention of the cecum by accumulating intracolonic gases from left to right towards the cecum. Let's also mention the Blumberg maneuver, which consists of causing pain in the right iliac fossa by suddenly releasing the pressure previously exerted with the hand in that area. There are also other maneuvers whose purpose is to contracture the psoas muscle, lifting the right lower limb in a rigid position, as well as, on the contrary, making the thigh flex strongly over the belly.

The cutaneous hyperesthesia produced by rubbing the skin with the nail, the muscular defense in the region and the pain caused constitute the classic Dieulafoy triad of acute appendicitis.

The pain caused suggests first of all acute or subacute appendicitis and also of typhlitis, perityphlitis, adnexitis, plexitis, ileitis, psoitis, as well as an inflamed Meckel's diverticulum. Don't forget-

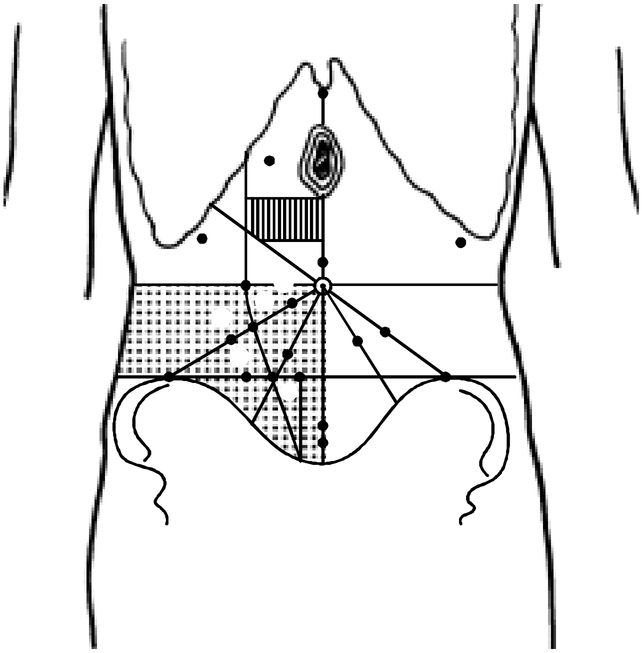


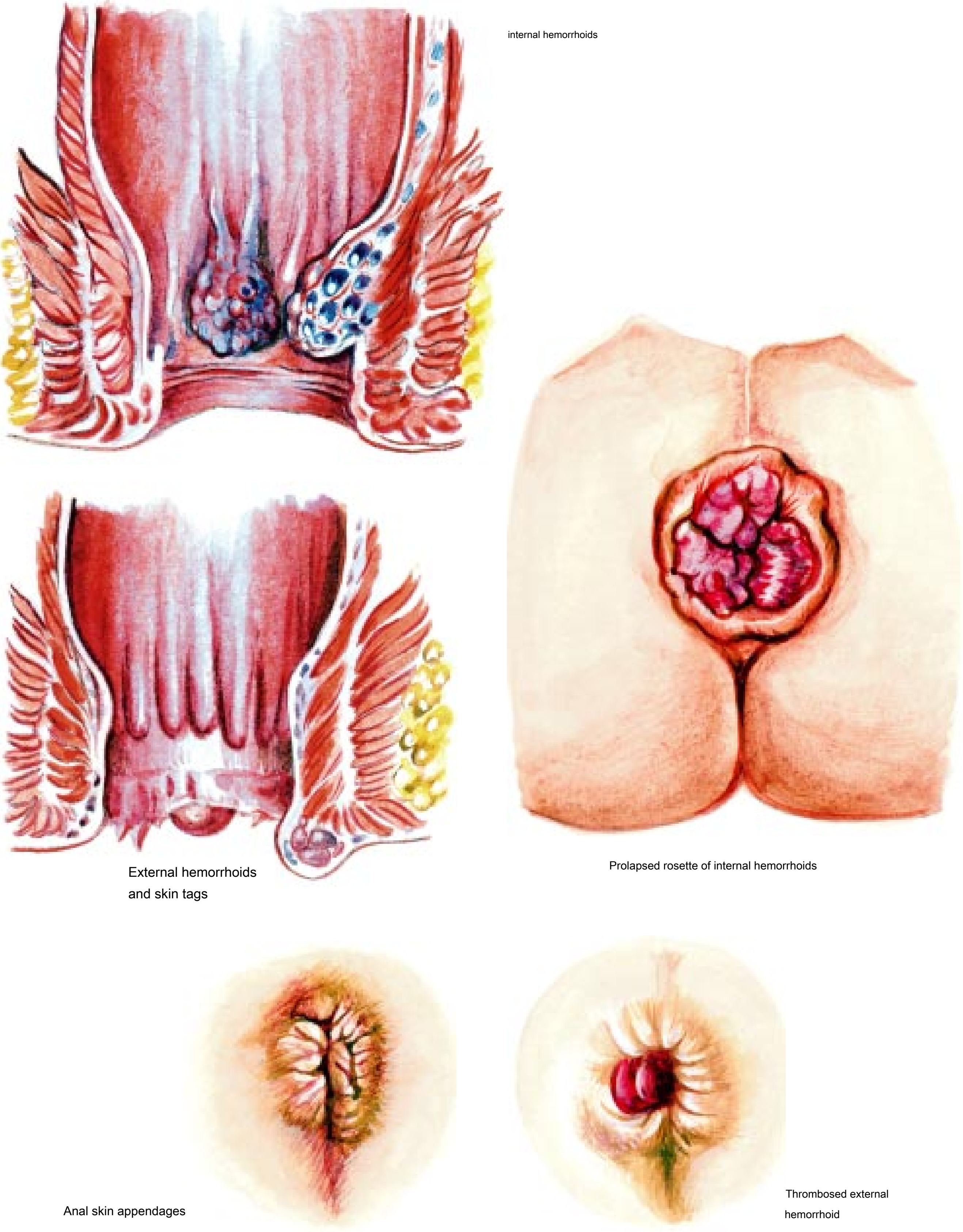
Fig. 61.26 Painful points and areas of the abdomen, particularly the appendicular area, according to Saint' Angelo: a, Morris; b, Monro; c, Mc Burney; d, Sonnerburg; e, Lanz.

We note that depending on the constitutional type, the pain can move up or down in relation to the right iliac fossa. Let us remember the possibility of atypical appendicitis, which causes physical alterations in the left iliac fossa and in the right lumbar region (retrocecal appendicitis).

Let us now point out as another finding in the pathological palpation, the lumps in the region. Let us first note the tumors, both benign and malignant, of the cecum, since those of the ileum and cecal appendix are rarer. They also determine lumpomas, tuberculous granulomas, eosinophils and regional ileitis, whose differential diagnosis is not easy due to palpatory characteristics.

Finally, let us mention other palpatory alterations of the cecum. The child's own ileocecal intestinal intussusception, which appears as a palpable black pudding-shaped tumor and varies in size spontaneously or on successive palpations; dilations of the cecum, either due to obstruction located in the colon, or due to primary cecal ectasia, in which cases the " gurgling or sloshing sound produced by the mixture of liquid and gas in the cecal cavity can be seen.

Of course, to complete the semiological study of the right iliac fossa, the pathology of the psoas muscle itself, the right tube and ovary, the ureter and the iliac plexuses and vessels of said region must be taken into account.



internal hemorrhoids

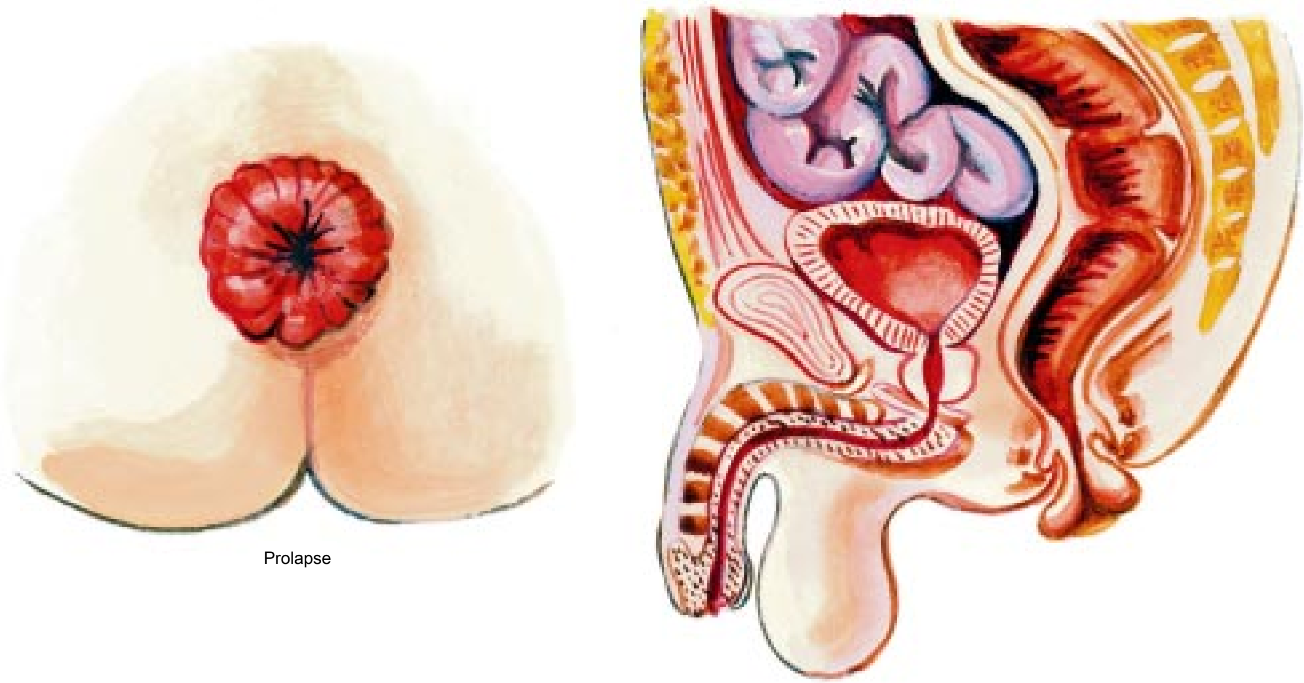
External hemorrhoids
and skin tags

Prolapsed rosette of internal hemorrhoids

Anal skin appendages

Thrombosed external
hemorrhoid

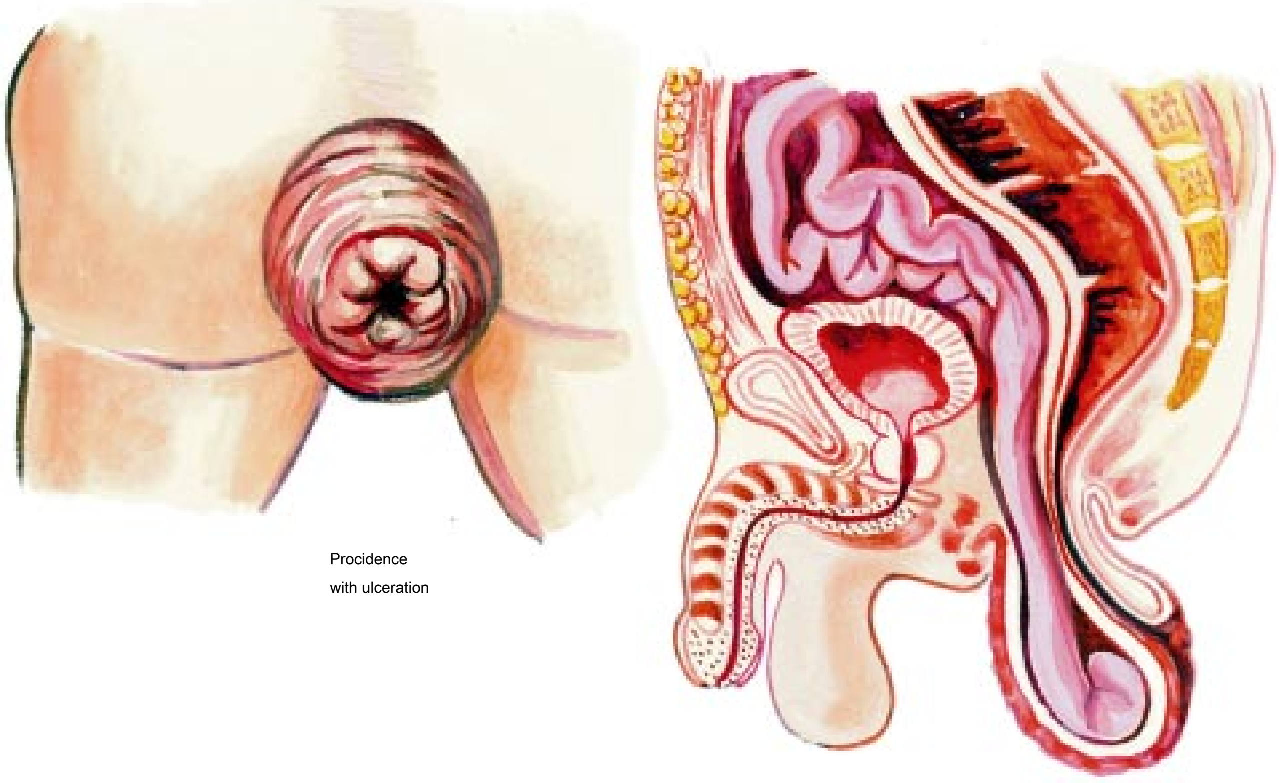
Fig. 61.27 Hemorrhoids.



Prolapse

Prolapse; only the rectal mucosa is affected

Procidence affecting all layers of the rectum: prolapse of the small intestine into the anterior rectal wall



Procidence
with ulceration

Fig. 61.28 Rectal prolapse.

EXAMINATION OF THE RECTUM AND ANUS

The anatomy, semitechnics and normal data of this segment were already discussed in volume 1, Section I, Chapter l6.

PATHOLOGICAL DATA

Inspection

1. Hemorrhoids (fig. 61.27). Those that in one way or another protrude into the anus. We must first consider the external ones, clearly visible especially if the patient makes a pushing movement, which appear as thick cords or globular formations, purplish or pink, painless, that surround the entire contour of the anus; Secondly, internal hemorrhoids, which can sometimes protrude. Hemorrhoids can be identified on inspection, with thrombosis, appearing turgid like bluish or blackish grapes. Sometimes their inactivity is appreciated and they are flaccid, fibrous, simulating hanging excrescences.

2. Fissure of the anus. It is nothing more than a regularly deep, triangular exulceration, oriented along the folds of the anus; It generally sits in the posterior or coccygeal angle of the anus, causing pain during defecation, especially in constipated people, and can bleed and become infected.
3. Fistula and abscesses. The external orifices of the fistulas and sometimes areas of swelling or true purulent collections are observed.
4. Prolapse. It occurs more frequently in children and the elderly. It consists of the exit through the anus of one or more layers of the rectum, like a cylinder (fig. 61.28).
   It can be partial or total; reducible or strangled.
5. Malignant tumors. Visible, such as epithelioma of the anus, which may appear ulcerated or nodular.
6. Other injuries of the anus. These can be condylomata acuminata, which appear as excrescences and are generally caused by leucorrhoea, gonorrhea, or parasitic or viral infections; flat, rounded, whitish condylomas, of a syphilitic nature, and benign tumors such as papilloma.

   Skin lesions are common and can coexist with other skin lesions on the body. Finally, let us also mention the scratching lesions of anal itching, whether idiopathic or secondary, eczema, etc.

rectal examination

By touch, what was indicated in the inspection is verified, and other useful details are added that contribute to the diagnosis:

1. Alterations in the tone of the anal sphincter. They are hypotonia and atony, called by some anal patulosis, which is found in the elderly and malnourished subjects, in rectal conditions, proctitis, hemorrhoids, etc. Anal hypertonia is also distinguished, very common in nervous patients, which makes the examination difficult. In general, it is present in all cases of lower rectal lesions near the anus, infected and painful, so it is necessary to use analgesic lubricants to overcome them.

2. Tumors of the rectum. Especially carcinoma; It is diagnosed in a high percentage only by touch, although rectoscopy with complementary biopsy is always recommended. Let us also point out fecalomas of the rectum, sometimes large, which constitute false tumors and are due to fecal impact." In turn, infiltrating tumors of the pelvic floor (cul-de-sac) are important, generally secondary to neoplasms of the upper abdomen (carcinoma of the stomach and pancreas), which is verified by touch (step sign).

3. Stenosis of the rectum. Produced, either by subacute lymphogranulomatosis or Nicolás and Favre disease, or by neoplastic origin, or by syphilitic or other etiology.
   Touch alone indicates the level, the length, whether it is circular, regular, irregular, active, etc., which guides its etiology.
4. Inflammatory processes of the lower abdomen corresponding to higher segments of the digestive tract. They are diagnosed through combined touch and palpation.
5. Foreign bodies. They can reach the rectum through accidental oral ingestion (fruit seeds, glass balls, buttons, etc.), or rectally, most often voluntarily (hair clips, candles, small bottles, etc.), used for autoerotic purposes.

In pathological cases we will perform anoscopy and rectosigmoidoscopy, which will allow us to better specify the lesion and obtain swabs, secretions or biopsy of the different lesions that we have studied.

DIGESTIVE SYSTEM. COMPLEMENTARY EXPLORATIONS

62

Introduction

It would not be too much of an exaggeration if we said that at the end of the 19th century or the beginning of the 20th, studies dedicated to complementary exploration practically did not exist or were reduced to a few short pages within the broad limits of a work on digestive semiology.

Today, on the contrary, we have been forced to make a synthesis of them because otherwise they would be endless, since any complementary exploration method, particularly studied, can constitute an extensive monograph.

Scientific-technical and technological progress has led to the availability of a whole series of imaging and endoscopic diagnostic procedures, as well as various examinations that allow studying the morphology and function of the organs of the digestive system. In no way can these modern techniques replace the history and physical examination. The examinations must be carried out based on a presumptive clinical diagnosis, and efforts will be made to indicate only the complementary tests that are essential to reach a positive or certain diagnosis.

Complementary exploration of the abdominal cavity is very useful and of great value in gastroenterological diagnosis.

For educational purposes we will group the exploration methods, and we will study some of them:

Paracentesis (ascitic puncture).
Complementary imaging exploration.
Endoscopic methods.
Exploratory methods by probing.

PARACENTESIS (ASCITIC PUNCTURE)

DEFINITION

It is an operation that consists of punctured the abdomen to evacuate a fluid collection from the peritoneal cavity or large intra-abdominal cysts.

INDICATIONS AND CONTRAINDICATIONS

It is indicated in all ascites, for diagnostic purposes: extraction of fluid for complementary examination, which includes cytology, post-puncture palpation, pneumoperitoneum, etc.; also, sometimes, for therapeutic purposes (evacuation of large ascites or large cysts). It is contraindicated in cases of multiple adhesions in the abdominal cavity.

MATERIAL

A trocar is needed, generally the medium one from the Potain collection (which come superimposed); The caliber is changed according to the variant of each patient. A thick needle is sometimes used, especially for minimal exploration or evacuation. Suitable bottles for laboratory studies and tanks are prepared to receive the liquid, as well as sterile and antisepsis material.

TECHNICAL PROCEDURE

We will describe the technique developed by Llanio, since it is very simple and easy to perform; Furthermore, the discomfort caused to the patient is minimal and allows partial or total evacuation of the abdomen slowly:

1. The patient is placed lying down or leaning slightly on the side that is going to be punctured. The puncture must be performed, if it is on the left side, at the midpoint of the line that joins the navel with the anterior superior iliac spine: if it is on the right side, at the same point or on the midline itself, in cases of operations in the right lower quadrant. On the right side, the following should be taken into account: the large hepatomegalies and the distended cecum; on the left side, the large splenomegaly and in the midline, the full bladder.

2. The selected area is sterilized (iodine, thimerosal and alcohol).

3. Local anesthesia is administered at the previously chosen point. To do this, a short needle, No. 26, one of those used to inject insulin (Luer size 26 G 1.56 cm), is inserted almost parallel to the skin (intradermal), and 1-2 mL of anesthetic is injected until a wheal is produced (fig. 62.1). The syringe is then brought to a vertical position and infiltrated as far as the needle can reach. Next we use a slightly larger and longer gauge needle (Luer size 22 G 3.75 cm, generally). The length of this needle depends on the thickness of the patient's abdominal wall; In this way, the infiltration of all planes is continued, performing aspiration from time to time, to avoid intravascular injection of the anesthetic (fig. 62.2).

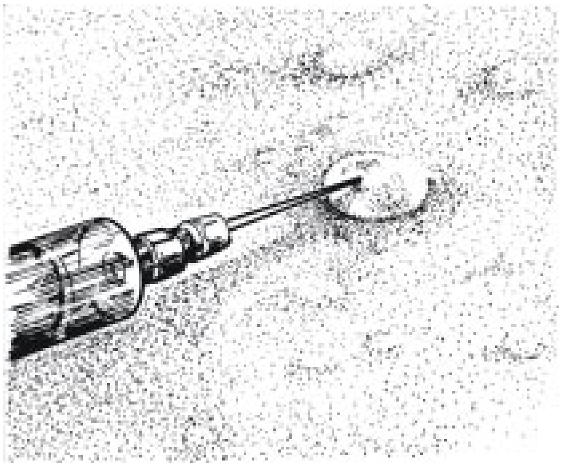


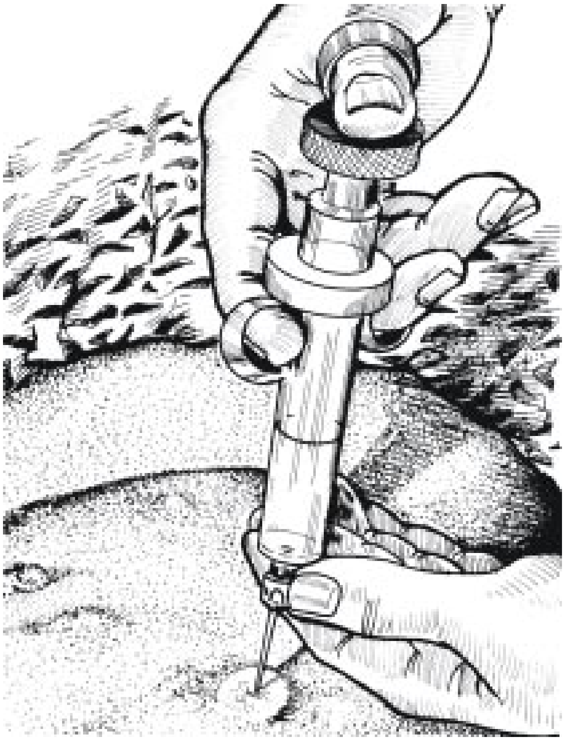
Fig. 62.1 Paracentesis. Intradermal infiltration of noVocaine.

We must keep in mind that it is necessary to adequately anesthetize the parietal peritoneum, since failure to do so may cause pain when passing the needle.

As anesthetic we use 1-2% lidocaine, ibecaine or novocaine, of which 10-20 mL are necessary.

4. An 18 or 16 gauge needle connected to an infusion set is inserted and once in the abdominal cavity, the liquid is allowed to spontaneously escape into a bottle (fig. 62.3). The needle is fixed to the skin.

Fig. 62.2 Paracentesis. Anesthesia infiltration in all planes of the wall.



5. This allows us to measure the amount of liquid that is being extracted and see its characteristics, as well as stopping the puncture when we estimate that its objective has been achieved (exploratory purposes for examination) or the patient's relief by reducing abdominal tension.
6. It is not necessary to give stitches, a swab with a tape is enough.
7. If the exit is stopped by an intervening intestinal loop, the needle is introduced to a greater or lesser depth or the direction is changed.

POSSIBLE INCIDENTS AND ACCIDENTS

Syncope or lipothymia, of a reflexogenic nature, is very rare. The puncture can be negative: due to the absence of fluid, when it does not fall into the peritoneal cavity, when the fluid is cysted, when there are adhesions, etc.

Let us point out the exvacuo hemorrhage, which is exceptional; In our opinion it is due to injury to a vessel in the abdominal wall. Peritoneal infection is very unlikely if performed with a minimum of asepsis.

COMPLEMENTARY EXPLORATION BY IMAGING

SIMPLE RADIOGRAPHY

Simple chest x-ray is ideal to demonstrate the presence of free air in the abdominal cavity (pneumoperitoneum) when perforation of the A hollow viscus and that of the abdomen is useful to assess the situation and size of the solid viscera (liver, spleen, kidneys), the presence of calcifications or lithiasis, free fluid in the abdominal cavity, ectopic air and above all for the study of the aerial pattern of the loops and the presence of air-fluid levels to define if there is an intestinal obstruction and at what level it is located.

CONTRAST RADIOGRAPHY

Barium contrast exams are used to evaluate the inside of the entire digestive tract. They allow the direct study of the mucosa and indirectly of the rest of the mural layers and extrinsic alterations.

ULTRASONOGRAPHY

Ultrasounds are nothing more than sound waves imperceptible to the human ear. The application of ultrasound in medical diagnosis is based on the principle of echo and for this reason this examination is also called ultrasound.

Ultrasonography is a non-invasive procedure that has the advantage of not using ionizing radiation.

It is the method of choice to show gallstones. It is a very efficient test for the diagnosis of liver tumors, as well as the primary method of choice in the morphological diagnosis of hepatobiliary diseases and frequently the only study necessary. Ultrasonography is superior to other modalities in differentiating cystic lesions from solid lesions and is very sensitive in detecting ascites. Ultrasound allows the study of the thickness of the walls of the gastrointestinal tract, which is useful in the diagnosis.

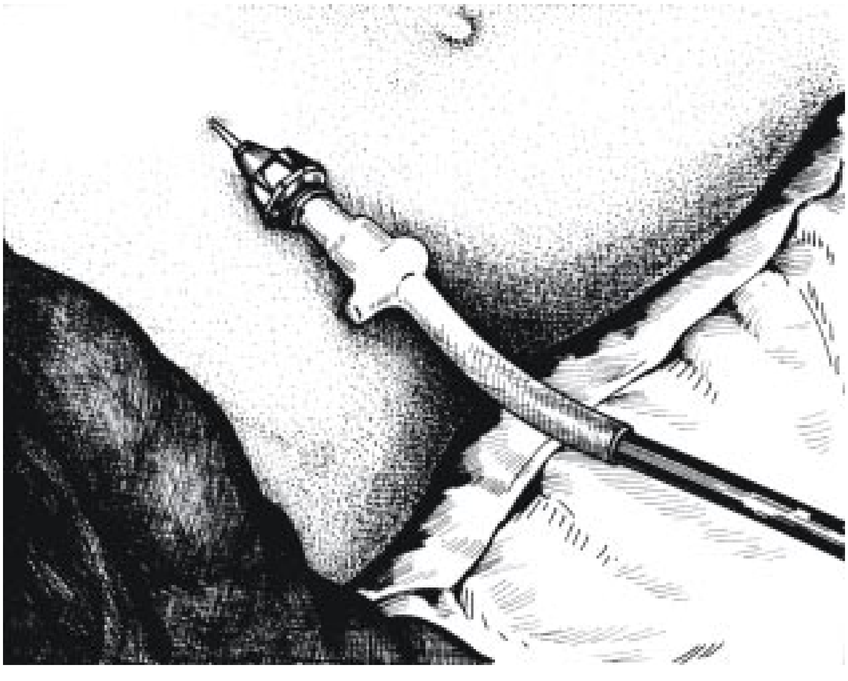


Fig. 62.3 Paracentesis. Agja placed in the abdominal cavity. Drainage of ascites.

diagnosis of inflammatory diseases (for example, Crohn's disease), ischemia, lymphoma and tumor lesions.

Percutaneous fine needle aspiration is possible for cytological diagnosis and drainage procedures of abscesses and cysts and with greater ease and lower cost than computed tomography.

Advances in ultrasonography allow the execution of intraoperative ultrasound studies, by applying a transducer to an organ exposed in abdominal surgery. The application of endoscopic ultrasound has also constituted an advance in this sense, by coupling the transducers to fiber optic endoscopes. This new imaging procedure shows the thickness of the wall of the esophagus, stomach and duodenum and identifies diffuse and focal intramural lesions. It is also useful for the diagnosis of early pancreatic lesions.

COMPUTERIZED AXIAL TOMOGRAPHY (CT)

This examination, which also uses X-rays, but not in the conventional way, allows the assessment of the digestive system, complementing barium studies and providing additional information. It makes it possible to study the digestive tract, inflammatory and tumor diseases of the pancreas, it is a method of choice for the diagnosis of liver tumors, it also allows the study of the gallbladder and bile ducts, as well as abdominal and retroperitoneal lymphadenopathy.

For the correct assessment of the mural and extramural component of the lesion and the visceral extraintestinal pathology, it is necessary to opacify and distend the gastrointestinal tract with oral water-soluble iodinated contrast, occasionally supplemented by the rectal and intravenous route.

Percutaneous fine needle aspiration of a tumor lesion for cytological study constitutes a complementary method to CT. This procedure allows the diagnosis of pancreatic cancer, primary and metastatic tumor of the liver, as well as tumor conditions of the lymph nodes.

Percutaneous drainage of an intra-abdominal abscess guided by CT is a therapeutic variant of this diagnostic procedure.

NUCLEAR MAGNETIC RESONANCE

Magnetic resonance imaging is a tomographic technique that guarantees magnificent image quality. Its use in the diagnosis of primary and metastatic liver tumors guarantees greater sensitivity when compared with computed tomography, ultrasonography and scintigraphy, but it has not been determined even if its specificity is greater. Very useful in the diagnosis of cavernous hemangiomas and their differentiation from other benign tumors. It allows obtaining images of the gallbladder, detecting cholelithiasis and differentiating concentrated from non-concentrated bile.

Current techniques allow intestinal motility to be evaluated by performing tomographic slices in hundredths of a second.

IMAGING WITH RADIOISOTOPES

It is possible to make the diagnosis of different diseases of the digestive system through the images obtained after the administration of various radioactive isotopes and study the scintigraphy obtained.

This technique has been used to study the gallbladder and bile ducts, to detect the presence of ectopic gastric mucosa, especially in Meckel's diverticulum and Barret's esophagus. Methods are available to detect sites of acute and chronic gastrointestinal bleeding. With radioisotope images it is possible to study gastric emptying and the existence of pathological gastroesophageal reflux.

Liver scintigraphy makes it possible to identify lesions that occupy space, such as tumor, abscess or hematoma, and evaluate hepatocellular conditions. The sensitivity for the detection of primary and metastatic liver tumors is comparable to that of computed tomography and ultrasonography.

ENDOSCOPIC METHODS

The development of fiberoptic endoscopy and videoendoscopy, which has occurred in recent decades, represents, without a doubt, one of the greatest advances in the diagnostic field of gastroenterology.

It is accepted that endoscopic examination constitutes the most sensitive procedure for the study of the upper digestive tract and colon.

Endoscopy can be performed with fiber optic equipment, which basically consists of a light source with a xenon or halogen lamp that transmits powerful light to the endoscope. Video endoscopy can be defined as endoscopic images transmitted to a television monitor. This can be obtained in two ways: by adapting a video camera to the proximal end of a fiber optic endoscope (indirect video endoscopy) or by electronic endoscope (direct video endoscopy). In this, the fiber optic system has been replaced by an optical electronic sensor that can be considered the heart of the system.

LAPAROSCOPY OR PERITONEOSCOPY

Definition

Laparoscopy (lapara: abdomen; skopein: observe) is the direct inspection of the abdominal and pelvic cavities by means of an optical instrument: the laparoscope, now using fiber optics to transmit light. Also under the same principle indicated above, videolaparoscopy is used. By this means, the peritoneal surfaces and the organs contained in the abdominopelvic cavity can be easily examined, which allows a quick and direct diagnosis of their alterations, as well as using this procedure to obtain samples for histological and cytological study, among others.

History

This test was reported for the first time by Kelling, in 1901, at the LXXIII Congress of German Physicians and Naturalists in Hamburg, demonstrating for the first time celioscopy in a dog. He later used the method on humans. However, from 1910 to 1914 Jacobeus published several works on laparoscopy and thoracoscopy and although he recognized its value in the diagnosis of certain conditions, he described it as brutal and aggressive, an opinion that somewhat hindered its development. However, in 1925, Nadeau and Kampmeier published an extensive review of the literature, citing the experiences of some 25 authors, from 1910 to 1925.

On the other hand, Kalk and Henning designed laparoscopes and carried out very interesting studies.

In our country, laparoscopy was introduced around 1932, by Núñez Portuondo, who performed some laparoscopies on his patients and those of other doctors, and as a culmination of them he published a work where he presented his results.

Starting in 1954, Llanio began to perform them and after 1959 he fully developed not only laparoscopy, but also complementary methods and created the Cuban School of Endoscopy, which has the greatest experience in the world.

Until 1956, this examination was considered contraindicated in cases of acute abdomen. It was on that date that emergency laparoscopy began to be performed in our country by Llanio, a pioneer of this diagnostic procedure. This author is also the founder of the only emergency laparoscopy service that exists in the world, located at the Institute of Gastroenterology, which provides emergency service 24 hours a day.

Applied anatomy and physiology

We will now describe the fundamental data of the abdominal cavity.

Anatomy

Boundaries. In its upper part it has the diaphragm; In the lower abdomen it is continuous with the pelvic cavity; In its lateral and anterior parts it is made up of the abdominal wall. All covered by the parietal peritoneum.

Content. For greater ease we must consider the contents of the abdominal cavity and the pelvic cavity.

1. Abdominal cavity (figs. 62.4 and 62.5):

   Organs of the digestive tract: abdominal esophagus, stomach, duodenum (mitra), jejunum, ileum, cecum, appendix and colon.

   Annexes to the digestive tract: liver and gallbladder.

   Other organs: spleen.

   Greater and lesser omentum, some mesos and ligaments, muscles, etc.
2. Pelvic cavity:

   Uterus, tubes and ovaries.

   Rectum and bladder.

   Psoas, ureter, iliac arteries and veins, inguinal fossae and ligaments.

Physiology

Normally all the organs are functioning, and the movements of the diaphragm can be appreciated, which are transmitted to the entire content; peristaltic contractions of the entire gastrointestinal tract and also of the ureter, especially the right one.

Indications

Laparoscopy can be used not only for diagnostic purposes, but also for therapeutic purposes.

For diagnostic purposes

1. All ascites, as well as lesions of the peritoneum. In reality, laparoscopy can constitute an extension of abdominal paracentesis.

   As Jacobeus has very well said, it would be an unforgivable and rude error not to take advantage of this contingency to verify, without a doubt, the cause of the spill, all the more so since it is not easy to know, most of the time, without using this procedure.
2. All acute medical liver diseases, except perhaps very benign hepatitis.
3. All the so-called "sequelae of hepatitis", if its cause outside the liver has not been determined by other diagnostic means.
4. All chronic liver diseases.
5. All cases where liver neoplasia is suspected. In addition, it allows a “biopsy” to be performed

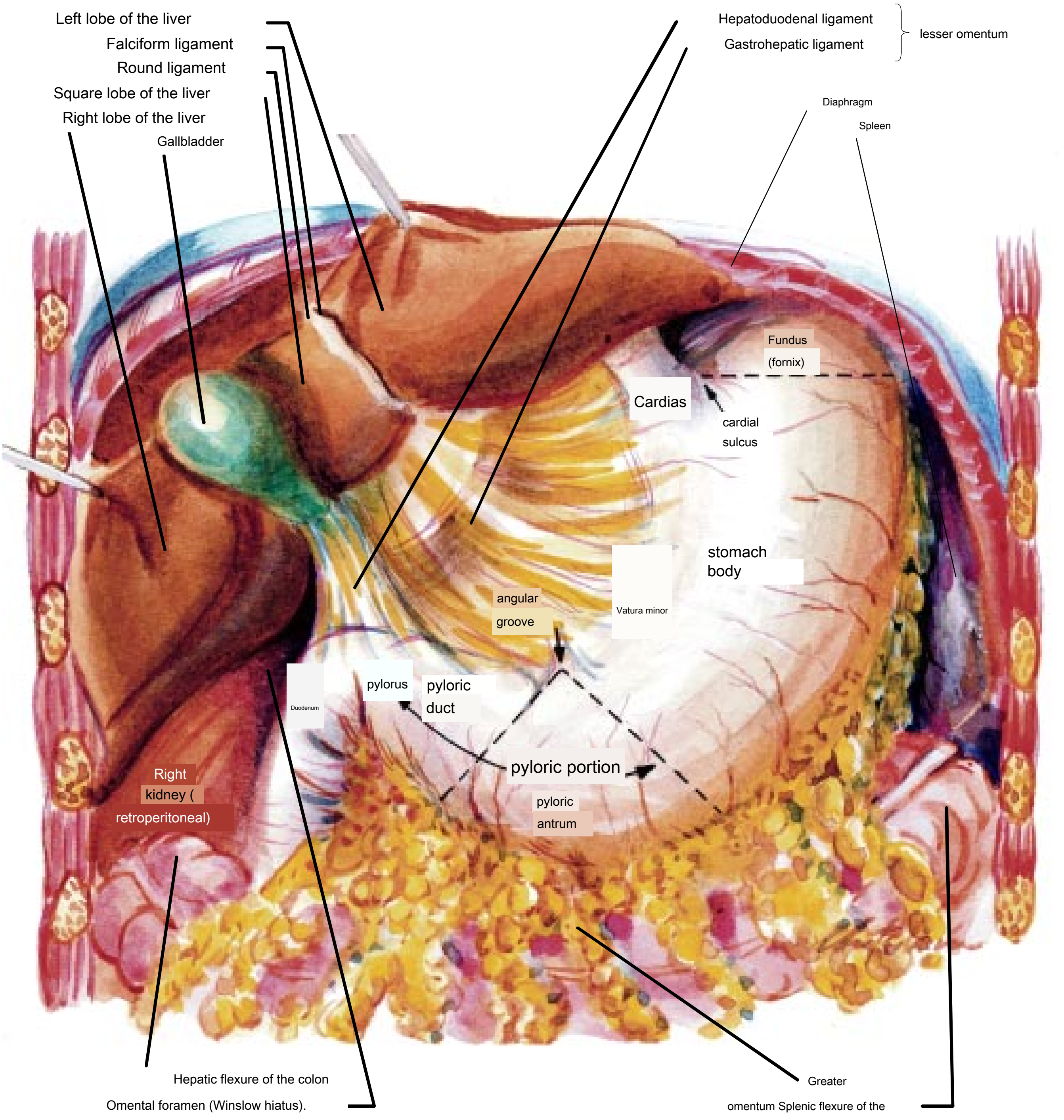

Left lobe of the liver
Falciform ligament
Round ligament
Square lobe of the liver
Right lobe of the liver
Gallbladder
Hepatoduodenal ligament
Gastrohepatic ligament
lesser omentum
Diaphragm
Spleen
Fundus
(fornix)
Cardias
cardial
sulcus
stomach
body
angular
groove
Vatura minor
Duodenum
pylorus
pyloric
duct
pyloric portion
pyloric
antrum
Right
kidney (
retroperitoneal)
Hepatic flexure of the colon
Omental foramen (Winslow hiatus).
Greater
omentum Splenic flexure of the

Fig. 62.4 Anatomy of the abdominal cavity.

directed "whose percentage of diagnosis is much higher than blind liver biopsy.

6. All obstructive jaundice, to determine whether it is a medical or surgical jaundice (with the help of cholangiography), its location and etiology.
7. Conditions of the gallbladder and bile ducts. In the direct visu examination we can diagnose the neoplasias and inflammatory processes of the gallbladder, among others.
8. Those cases of abdominal tumor syndromes, in order to define their nature and location.
9. Cases of diagnosed neoplasms, to know whether or not there are abdominal metastases, especially liver and peritoneal metastases, which can be very

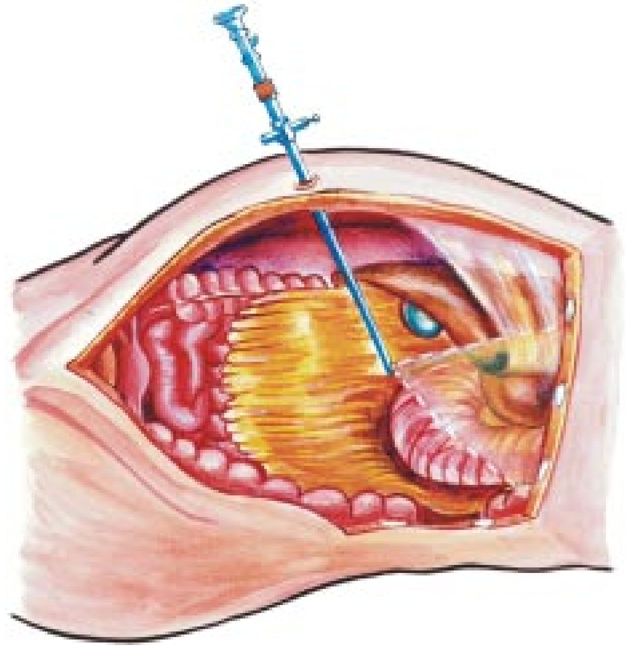


Fig. 62.5 Organs of the abdominal cavity visible by laparoscopy.

important when assessing whether or not to use surgical treatment.

10. To assess the stage and evolution of lymphomas.
11. Chronic abdominal pain syndromes, if after the remedies have been exhausted, their etiologies are not defined.

12, Acute abdominal syndromes, with greater or lesser peritoneal reaction, especially in those cases where the clinical or surgical diagnosis, and therefore the therapy to follow, is not defined (laparoscopy in emergencies).

13. Splenic conditions, especially splenomnegalia, infiltrative lesions, etc., require do its nature. Allows obtaining directed biopsy.
14. The pathological symptoms of the right iliac fossa, so frequent and so difficult to interpret.
15. Pelvic conditions, tumorous or not, where there are doubts or inaccuracies in the diagnosis. It should not be forgotten that in cases of suspected extrauterine pregnancy it is the best diagnostic method.

For therapeutic purposes

Initially, the section of adhesions was performed through laparoscopy as a treatment for adhesion syndrome and electrocoagulation and section of the tubes for the sterilization of the woman. Nowadays, almost all abdominal surgical interventions that are done by conventional surgery are performed through laparoscopy, for example, cholecystectomy, splenectomy, hysterectomy, hemicolectomy, appendectomy, ectopic pregnancy, etc.

Contraindications

1. Let us point out that a few years ago acute abdominal processes (cholecystitis, appendicitis, etc.) were considered a formal contraindication to this examination. due to the danger of spreading the infection. However, Llanio has demonstrated its safety and usefulness especially in those acute or subacute cases of the abdomen, where it has not been possible to specify the surgical or medical conduct and also in those where, even accepting that it is surgical, there is no precise idea of the site of origin of the injury, and therefore, this pre-operative examination can decide the location of the process and, consequently, the surgical incision.

2. When there are extensive areas of adhesions, its use is dangerous. In these cases, if it is essential to perform laparoscopy, it should be done at a site quite far from the surgical area. Taking this precaution, there will be no problems when doing it.
3. It should be avoided in tuberculous, adhesive peritonitis, because a fixed loop can easily be perforated. However, when it is in the exudative phase with ascites, it can be performed and serves not only to make the diagnosis but also as treatment.
4. In decompensated cardiac patients without ascites and in chronic pulmonary patients with marked respiratory failure, since introducing air into the abdominal cavity in a certain quantity causes an elevation of the diaphragm, with a greater decrease in respiratory capacity.

   In special cases and with care it can be carried out, especially if they have ascites, since by replacing the liquid with air, the patient's respiratory conditions do not worsen.
5. Finally, great care must be taken in large tumors, especially if adhesions are suspected in the anterior wall.

Normal data

We will briefly describe here all the formations (organs, vessels, omentum, etc.) that can be visualized with laparoscopy (see figs. 62.4 and 62.5):

1. First, the entire anterior and lateral parietal peritoneum, the greater omentum and some mesos, also the diaphragm and the phrenic center, as well as the heartbeats transmitted to it, are observed.
2. Among the organs of the digestive tract, the liver is easily visible, on its anterosuperior side and part of the lower side, of both lobes, with its red color

characteristic brownish color and its smooth surface, the round ligament that comes from the umbilicus and the suspensory ligament.

3. The gallbladder is seen to be bluish green when it is full. Also the stomach, especially the anterior surface of the body and the antrum. The greater curvature, with its vessels, and sometimes, the lesser omentum, with peristaltic contractions being appreciated in said organ. Sometimes the abdominal esophagus is visible.

4. In the left hypochondrium, the spleen is seen, red in color and with a smooth surface.
5. The colon is also visible, in almost its entire length and its different segments, the greater omentum and the loops of the small intestine with their peristaltic movements. Of special interest is the right iliac fossa, where the cecum, terminal ileum and appendix can be perfectly seen in most of the cases.

cases.

6. Other organs and structures, such as the uterus and adnexa (tubes and ovaries) are clearly observed, as well as the psoas and iliac vessels, the inguinal fossae, the ureters, especially the right one, and the bladder.
7. Retroperitoneal formations are not visible in the normal state, but it is possible to visualize the head and body of the pancreas.

Pathological data

For educational purposes we will begin this description with the injuries that affect:

- Peritoneum.
- Liver.
- Gallbladder.

Stomach.
- Jejunum and ileum.
- Large intestine.
- Spleen

Retroperitoneal organs and formations.

We will not refer to genital alterations, because although they provide important data through this endoscopic method, they completely go beyond the limits of complementary digestive exploration.

Peritoneum

Fundamentally three types of lection can be seen.
sions:

- Peritoneal tuberculosis.
- Neoplastic peritoneal seedings (peritoneal carcinomatosis).
- Adhesions (perivisceritis) of the organs to each other and from these to the abdominal wall and omentum.

Peritoneal tuberculosis, almost always of the type of yellowish-white miliary granules, which can affect an area or generalize. Other types of granulomas with very similar characteristics can also be observed.

Neoplastic peritoneal seedings (peritoneal carcinomatosis) have a similar appearance to the previous one, but are larger and more flattened.

Adhesions (perivisceritis) of the organs to each other, and of these, to the abdominal wall and omentum, are almost always postoperative, but in some cases, certainly more frequent than is thought, we have found multiple adhesions in non-operated bellies that correspond to primary perivisceritis, almost always of inflammatory etiology that has gone unnoticed (such as T.B.), or are due to blood coming from follicles. hemorrhagic. In some cases they are very vascularized, which makes their endoscopic section difficult.

These adhesions are responsible for many vague painful conditions in the abdomen.

Liver

The injuries that we most frequently observe in this organ are:

-Hepatitis.
- Cirrhosis.
- Neoplasms.
-Fatty infiltration.

Congestive liver of heart failure.
- Other injuries.

We make it clear that in its laparoscopic appearance, hepatitis depends on the evolutionary stage or its intensity. Thus we have the following stages or phases:

a) Acute hepatitis. The liver appears enlarged and bright red in color.
b) Severe hepatitis. We should point out that some acute hepatitis of great intensity, which we designate as acute or subacute necrosis of the liver, presents a condition in which the massive destruction of one or part of the two lobes predominates and whose laparoscopic expression is the increasing collapse of the liver surface with multiple regeneration nodules and with the appearance of a bombarded field, as Kalk has pointed out.

c) Persistent chronic hepatitis. The liver has a pale red color, the capsule loses its transparency and whitish tracts are observed.

d) Chronic active (aggressive) hepatitis. The liver is enlarged, pale red in color, the surface has a tendency to curl and sometimes presents micronodules; In addition, increased superficial vascularization and alterations of its border are observed.

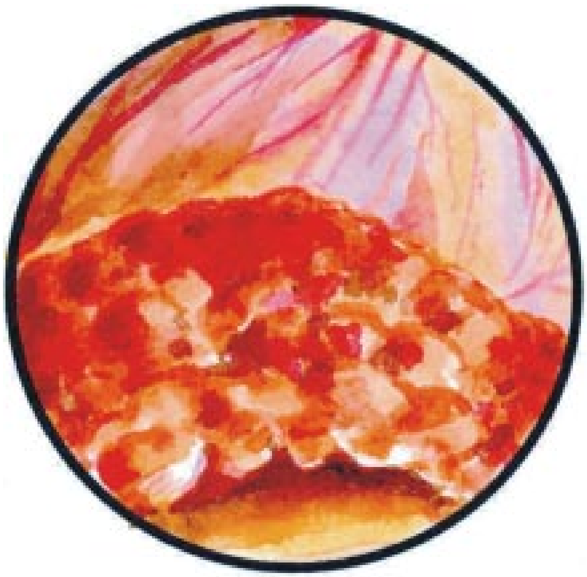
Cirrhosis deserves some preliminary considerations; Sometimes the surface of the cirrhotic liver appears discreetly wavy, of a paler or yellowish color and, above all, of increased consistency on laparoscopic palpation or puncture. Other times, however, the surface is frankly irregular, it can take on three fundamental types: the small nodule (3-5 mm), the medium nodule (10-20 mm) and the large nodules, like lemons or potatoes, which can be confused with malignant neoplasms.

On the other hand, the liver, according to the evolutionary stage of cirrhosis or the type of cirrhosis, has a variable size: larger, equal to or smaller than normal, sometimes almost atrophic. Furthermore, it may predominate over one lobe more than the other, although in cirrhosis the entire liver is always involved (Fig. 62.6).

We have already analyzed, by way of introduction, some general characteristics, especially regarding size, consistency, surface, etc. Let us now go on to specify the most salient characteristics of the most common types of cirrhosis in the clinic. So we have:

a) Portal cirrhosis, Laennec type (septal cirrhosis). In the early stages of its evolution, the liver may be large, with a slightly irregular surface and a paler color than normal. On the contrary, in the advanced stages, the liver is small, atrophic, its entire surface is covered with small nodules, queños, uniform and universally distributed; Its edge is thin, sharp, and its consistency is hard, fibrous, these being the most salient facts. It is also accompanied by a great vascularization, with a notable increase in caliber, especially of the veins, much better visible at the level of the suspensory and round ligaments (in the latter they frequently form true internal "jellyfish heads", around the navel, much more frequent than the external ones), as well as the greater curvature of the stomach, omentum, etc.

Fig. 62.6 Laparoscopic view of cirrhosis.



b) Postnecrotic cirrhosis. Almost always secondary to acute hepatitis or a toxic or degenerative lesion. It can adopt three laparoscopic appearances according to the size of its adenomas or nodules, which are: small nodules, medium nodules and large nodules, as previously described. The color of these livers is very similar to those of portal cirrhosis, although sometimes it is somewhat darker.

c) Biliary cirrhosis (Hanot type), It is mainly due to lesions that obstruct the extrahepaic bile ducts, especially if they are benign and are accompanied by added infection (cholangitis): but also caused (to a lesser extent) by obstructive lesions of the intrahepatic bile ducts. Whatever its mechanism of production, it appears as a large liver, olive green in color (not as intense as that of obstructive jaundice), with a finely granular surface, like that of a strawberry, or only discreetly undulating (Fig. 62. 7).

As a common corollary to all the forms of cirrhosis described, we can say that all these livers have great consistency (firm, hard) and that they are always accompanied by splenomegaly, more or less intense, with greater or lesser repercussions on the portal venous system.

Liver neoplasms can be secondary (metastatic), the most frequent, whose characteristic appearance almost requires no description. Let's just say that they appear as nodules of variable size, between a lentil and an orange, that are prominent on the surface with a small umbilication in their center, yellowish white in color and distributed capriciously in part or all of the viscera, and the site of origin can be somewhat oriented, according to its predominant distribution in the left or right lobe (fig. 62.8).

Primary neoplasms, on the other hand, when they adopt the multinodular form are quite close to the description of the previous ones, but do not present umbilication, and on the contrary are frequently acuminate, which serves to differentiate them.

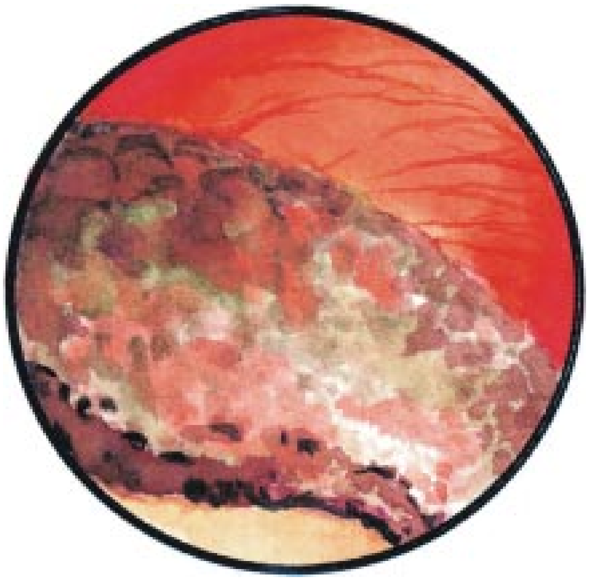
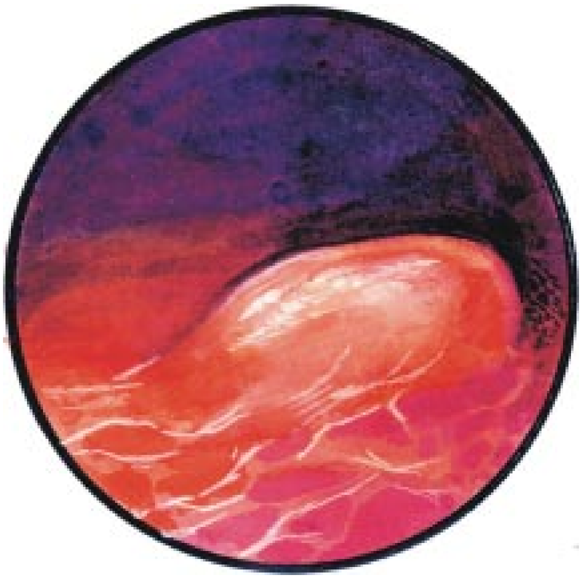


Fig. 62.7 Biliary cirrhosis.

On other occasions, let us point out that both one and the other, when they are located more deeply without reaching the surface, but close to it, can often be recognized by the detail that we have observed several times, which consists of a striking decrease in the intensity of the color of the surface of the liver that covers the neoplasm, compared to the rest. In these cases we have performed a puncture biopsy and have obtained, under the first layer of normal liver, a neoplastic fragment that has allowed the diagnosis.

In fatty infiltration, the liver appears noticeably enlarged, with a smooth surface; Its color is red, with a yellowish background (actually orange).

Fig. 62.8 Secondary neoplasia.



The congestive liver of heart failure can appear greatly enlarged, depending on the degree of insufficiency, with a red, sometimes purplish color due to blood stasis.

Other lesions such as sarcoidosis, tuberculosis, brucellosis and other granulomatosis can also be observed, which frequently give the liver surface a typical appearance of spots, which have a variable size of 1-2 mm and a whitish or yellowish color. In addition, liver abscesses are observed, generally single, yellow in color with great inflammation around them and frequent adhesions to the neighboring wall; the larva Migrans visceralis, which appears as more or less tortuous linear formations of yellowish color, which are prominent in Glisson's capsule. Finally, we have anomalies, such as abnormal segmentations of the lobes (polylobed liver) or cysts (polycystic liver), which are perfectly recognizable in this examination.

Gallbladder

The most frequent lesions that we can observe in this organ are: neoplasias, cholecystitis and vesicular lobulations.

Neoplasms are visible above all when they settle at the bottom or in the body of the gallbladder. They have a yellowish white color. Almost always in the most advanced periods they invade the hepatic bed by contiguity

Acute cholecystitis is the most expressive. They show a reddish color with great vascularization and often adhesions of the omentum to the gallbladder, forming a true plastron.

Vesicular lobulations. Phrygian cap gallbladder and double gallbladder are easily diagnosed.

Stomach

Here the lesions can be: neoplasms and ulcers.

Stomach neoplasms can be observed, mainly when they are located in areas accessible to the laparoscope and whenever they invade the external (peritoneal) surface.

Observable gastric ulcers are those that have the characteristics indicated in neoplasms. Let us state that some authors have devised a means of transillumination of the stomach (also of the sigmoid rectoyel) that allows these processes to be much better visualized. We almost always see the peritoneal surface dull or infiltrated by these lesions.

We can say the same about the duodenum at the level of the miter, when it is the site of ulcers on the anterior surface.

Jejunum and ileum

Inflammatory or neoplastic tumors and terminal ileitis (Crohn's disease) can be accurately diagnosed.

Rocavilla, among other authors, has greatly insisted on the usefulness of laparoscopy in the diagnosis of these inflammatory or neoplastic tumors, which are quite difficult by other methods.

We have verified the value of laparoscopy in cases of terminal ileitis or Crohn's disease and we have appreciated that the last loops appear infiltrated and rigid and that the peristaltic movements at that level largely stopped and only some of low intensity passed.

Large intestine

Here we can see: neoplasms, diverticula and the appendix.

The neoplasms are accessible to the eye, especially if they produce narrowings or protrusions on the surface, since the ulcerous or internal vegetative forms are not visible.

Diverticula appear as small pockets or projections of the intestinal wall.

The appendix deserves special attention. It is easily visible in most cases (except in certain anatomical positions) . When it presents inflammatory pathology, not only does its caliber increase and appear rigid, but its vascularization also multiplies; The greater omentum is frequently attached to the appendix.

Spleen

The lesions that can be diagnosed in this organ they are not:

Splenitis. Almost always concomitant in acute and chronic hepatitis, as well as in various systemic infectious processes. In these cases, the spleen appears double or triple its normal size and with a characteristic bright red color.

Granulomatosis (sarcoidosis, tuberculosis, granulias, Hodgkin, etc.). In these conditions, fine yellowish-white granules appear on the surface of the spleen. A very similar picture is observed in disseminated lupus erythematosus.

Fibrocongestive splenomegaly. In these conditions the spleen appears more or less large, with a typical violet, cyanotic color. In some clinical forms of cirrhosis, it acquires an extraordinary size, these are called Banti syndromes.

Other splenomegaly. Among those that cause the greatest enlargement of the spleen, we can mention leukemias, especially chronic leukemias, as well as lipid thesaurismoses of the Gaucher, Nieman-Pick and Hand-Schuller-Christian type. Also in amyloidosis it acquires a large size and a matte pink color in the infiltrated areas.

Retroperitoneal organs and formations

The processes located at this level, such as the pancreas, lymph nodes and great vessels, can only be suspected or diagnosed when they acquire a certain size and reject or lift the organs that cover them, or when they make their way between them, becoming directly visible and accessible to the biopsy. Thus we have been able to accurately diagnose tumors and especially cysts of the pancreas, sacciform aneurysms of the aorta and also mesenteric lipomatosis (Whipple's disease). If certain positions or accessory instruments are used, we can see the pancreas and take a biopsy of it.

Complementary examinations that can be performed through laparoscopy

We have already pointed out in the course of this chapter that there are complementary tests that can be performed through laparoscopy that complement it, such as:

Directed biopsy.

-Laparoscopic cholangiography.

Splenoportography with manometry.

- Cystography (cystography).

Laparophotography.

- Endoscopic surgery.

Targeted biopsy

Biopsy directed by laparoscopy has the advantage that it allows the site to be selected according to the macroscopic impression, in addition to being able to evaluate the macroscopic and microscopic characteristics of the organ (usually liver). to make the diagnosis.

Targeted liver biopsy can be performed by two methods: by trocar puncture and by punching with forceps. In trocar puncture we generally use the Menghini trocar or the universal Vin-Silverman trocar.

We also point out that it can be performed on other solid organs such as the spleen, as well as on tumors, whose solidity we check with the exploration needle.

In those places where the lesion is located in the parietal or visceral peritoneum (peritonitis, tuberculosis, sarcoidosis, etc.), we use the punch with special forceps designed for this purpose, which allows great diagnostic accuracy of these lesions.

Laparoscopic cholangiography

Laparoscopic cholangiography is an endoscopic-radiological technique. It has its precise indication in the diagnosis of obstructive jaundice, when it has not been possible to opacify the bile ducts through endoscopic cholangiography. It consists of puncturing the gallbladder with a special needle (under laparoscopic vision) and injecting a radiopaque iodinated substance (we use 50% biligrafin). Through this technique, visualization of the gallbladder as well as the intra- and extrahepatic bile ducts is achieved.

The careful study of the radiographs of obstructive jaundice (cholestasis) in which we use the stalking technique, provides us with the following information:

a) Topographic diagnosis of the obstruction site (fig. 62.9).

b) Diagnosis of the degree of obstruction (complete or incomplete)

c) Degree of dilation above the obstacle (fig. 62.10).

d) Etiological diagnosis (lithiasis, neoplasms) (figs. 62.11 and 62. 12).

e) Indemnity of the bile ducts in intrahepatic cholestasis (fig. 62. 13).

Splenography with manometry

Splenoportography with manometry allows us to know, on the one hand, the pressure existing in the portal circuit (manometry) and detect cases of hypertension and once this information is obtained, proceed to the injection of iodinated substance to study the splenoportal tree. Thus, the site of the partial obstruction that causes said portal hypertension can be verified, whether splenic, portal or intrahepatic, due to cirrhosis.

Cystography (cystography)

On some occasions when visualizing cysts, especially pancreatic, we have proceeded to puncture them (analysis of the content) and subsequently inject contrast, allowing us to know, through the x-rays, the exact location and approximate size.

Laparophotography

Especially in colors, taken through the laparoscope. It allows obtaining images that reproduce the lesions quite faithfully. This has two primary advantages: that of being able to judge the image by other doctors who did not attend the examination and that of representing a teaching method, since it serves to preserve it as an archival document, especially in many cases that heal later or in other cases. It serves as an evolutionary comparison (hepatitis and cirrhosis) and also in those where, for special reasons, the patient dies outside of hospital centers or where it is impossible to perform an autopsy.

Endoscopic surgery

Laparoscopy allows different surgical procedures to be performed, such as sectioning adhesions, sectioning or ligation of the tubes, biopsy of the ovary, puncture of small ovarian cysts, etc. In this way, surgical interventions and the discomfort or complications that they may bring are avoided.

UPPER GASTROINTESTINAL ENDOSCOPY

Through this endoscopic procedure, the esophagus, stomach and duodenum can be explored up to its descending portion, either separately (esophagoscopy, gastroscopy and duodenoscopy) or together (esophagogastro-duodenoscopy or panendoscopy). The entire mucosal surface is observed and samples can be taken for histological and cytological studies, photographs and videotapes can be taken. Like laparoscopy, upper endoscopy is performed for diagnostic and therapeutic purposes.

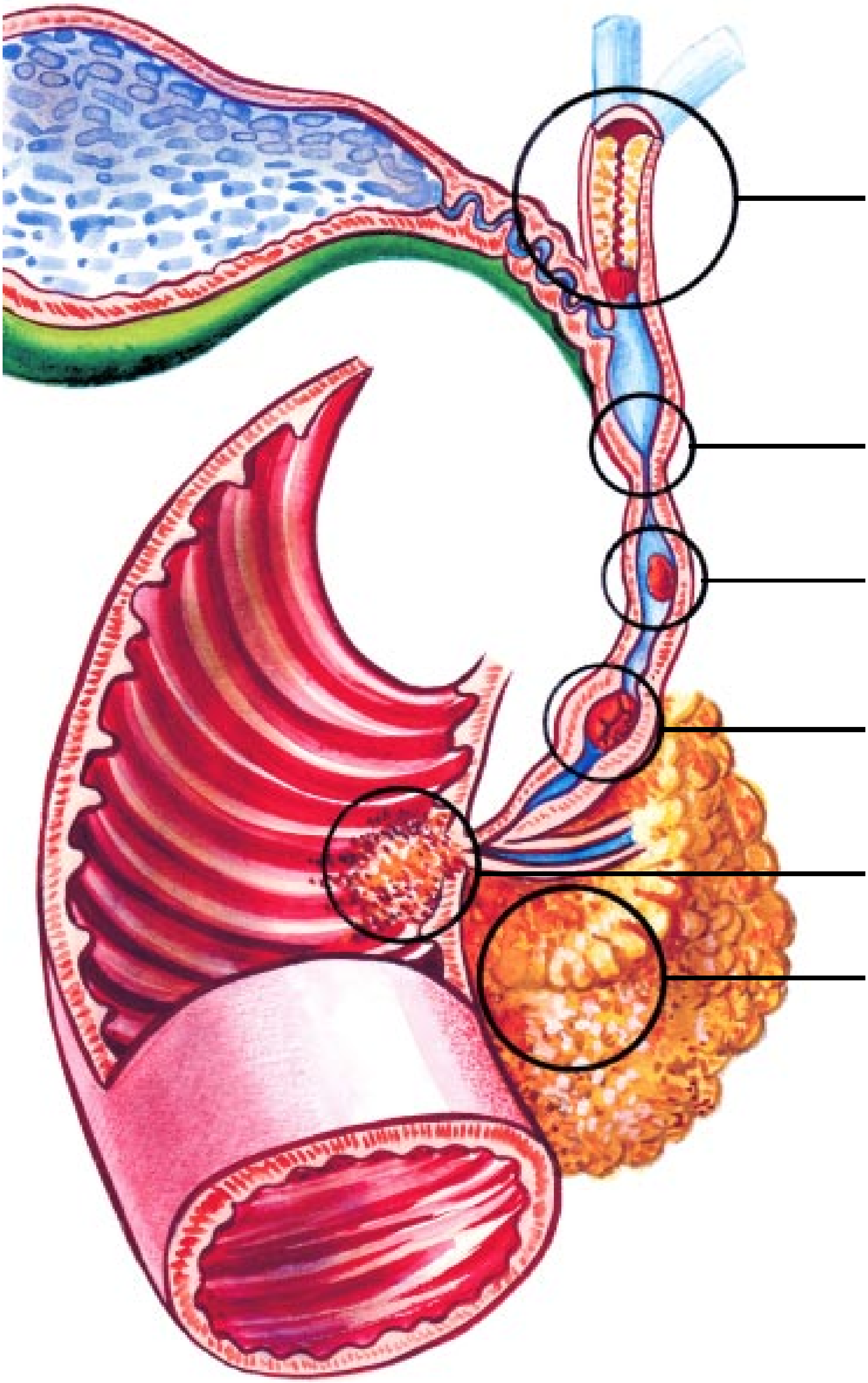
Esophagoscopy

Concept and importance

It is a simple optical method, through which we can perform a satisfactory inspection of the entire interior of the esophagus, from the hypopharynx to the cardia.
This examination can be performed with open or closed, rigid or semi-rigid tubes, which are inserted through the mouth, although for many years fully flexible fiber optic equipment that is practically harmless and painless for the patient has been preferably used.

Indications for diagnostic purposes

1. When any swallowing disorder suggests an injury to the organ (cancer, spasm, etc.).
2. To confirm the existence of an esophageal lesion previously suspected based on symptoms and radiology and, in addition, to help identify it through biopsy and cytological study.
3. In cases of persistent retrosternal pain or discomfort (peptic, caustic, infectious and drug esophagitis, hiatal hernia).
4. In cases of unexplained hemoptysis or hematemesis.
5. In the diagnosis of esophageal varices.



CARCINOMA OF THE LIVER DUCT.

If the tumor is located at this level the obstruction is complete, if it is located above, in one of the bile ducts terminals, the obstruction is incomplete.

STENOSIS OF THE COMMON DUCT0.

It is the result of surgical manipulation or inflammation of neighboring

GALLSTONE.

The obstruction is usually intermittent if the stone is not wedged.

LOCKED GALLSTONE.

The obstruction is complete due to edema and spasm of the common bile duct.

CARCINOMA OF THE AMPULA OF VATER.

It is the common example of an intrinsic tumor, generally malignant, that obstructs the passage of bile to the

CARCINOMA OF THE HEAD OF THE PANCREAS.

Important example of obstruction of the common bile duct due to the pressure exerted by an extrinsic injury.

Fig. 62.9 Etiological diagnosis of extrahepatic obstruction.

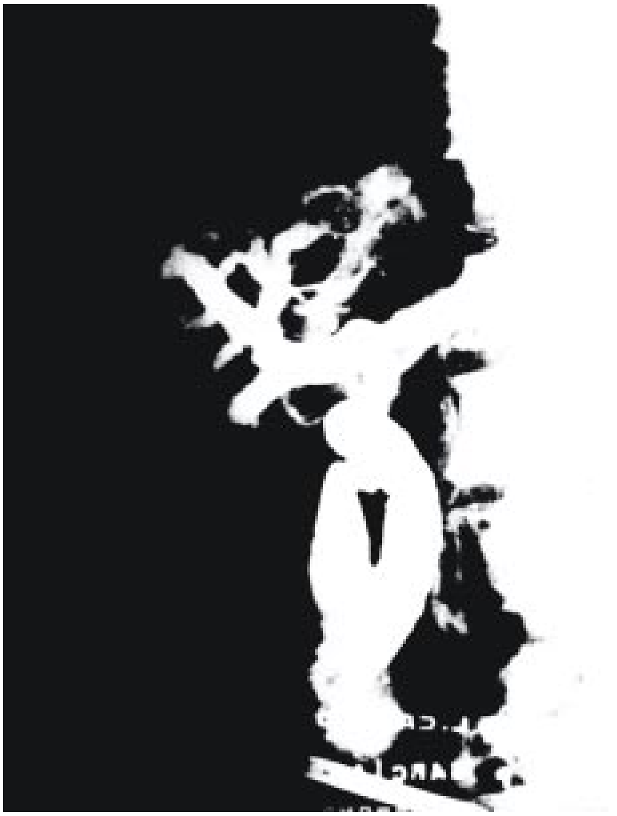
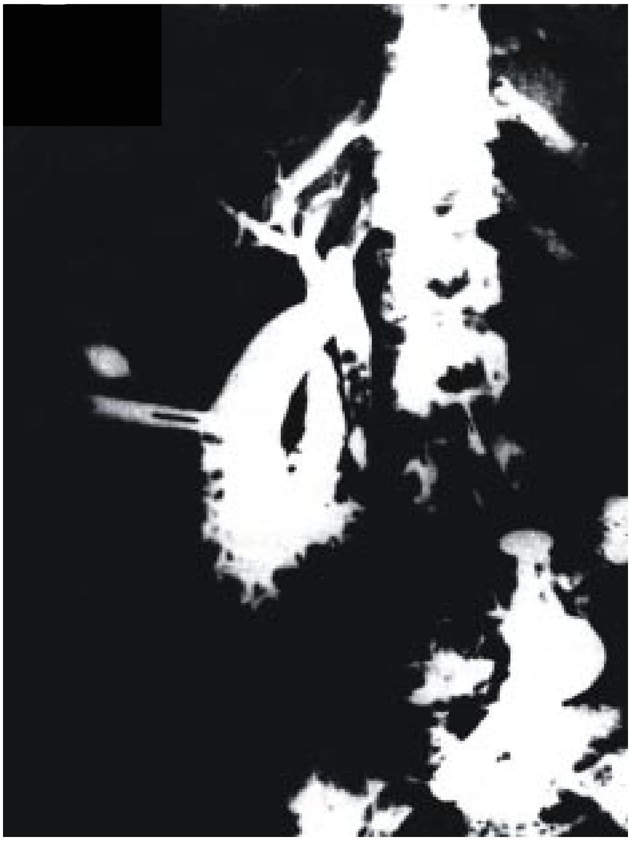


Fig. 62.10 Dilation of the bile ducts.

Fig. 62.11 Vesicular and common bile duct stones.



Indications for therapeutic purposes

1. As a safe mechanism for the dilation of stenosis of the organ, whatever its etiology.
2. For the treatment of many esophageal injuries, such as esophagitis, ulcerations, varicose veins, electrocoagulation of tumors, etc.
3. Palliative treatment of esophageal cancer by placing endoprostheses, applying laser therapy or absolute alcohol.
4. For the extraction of foreign bodies lodged or detained in the organ.

Contraindications

They practically do not exist, although care must be taken in advanced cachectic patients and in cases of aortic aneurysm. It should also not be attempted in the first days following burns of the esophagus due to ingestion of caustics (corrosive esophagitis due to hydrochloric acid or lyes).

Equipment (esophagoscopes)

Rigid esophagoscope. It consists of a rigid tube open at both ends, provided with its own lighting system, placed at the distal end, which is the one that is inserted. The esophagoscope (Fig. 62.14) is actually a speculum (an esophageal speculum) and is preferably used to remove foreign bodies.

Esophagofibroscope. As mentioned, this flexible equipment has almost completely displaced the previous type due to its safety and easy handling and for allowing, practically without discomfort for the patient, diagnosis, taking biopsies, cytology, extraction of foreign bodies, etc. (fig. 62.15).

Technical procedure

After sedating the patient, the esophagofibroscope is introduced; From the beginning, the mucosa, narrowings or lesions, etc. are observed, all interesting aspects are studied and it is easily removed.

This examination lasts only a few minutes and is perfectly tolerated, which is why it can be repeated in the evolutionary evaluation of certain conditions.

Normal data

The lumen of the esophagus is virtual and only appears with the progression of the esophagoscope. At the level of the esophageal mouth it is a transverse slit with two lips, anterior and posterior, red and applied to each other. The path is more or less straight, with moderate longitudinal folds up to the cardia. At the level of the diaphragmatic narrowing, the lumen narrows and appears in the form of two mucosal rims with thick folds or in a radiated form with a transverse groove between them.

The appearance of the interior of the esophagus seen with the esophagoscope is that of longitudinal folds that separate, are depressible and disappear when the tube passes and is compressed by it. The esophageal mucosa is generally pale pink.

Pathological data

As occurs in any endoscopic procedure, it is not easy to describe the multiple pathological images that are observed; However, modern equipment makes it easy to obtain very clear photographs of the lesions. The most frequent are:

- Narrowings.
- Dilations.
- Parcel expansions.
- Neoformations.

Areas of substance loss or ulcerations of the esophageal mucosa.

Changes in color and appearance of the mucosa.

- Foreign bodies.
- Vascular alterations.

Straits

Characterized by an evident decrease in the caliber or lumen of the esophagus, at which level the tube stops and cannot progress easily. They must be distinguished:

In the spastic type, in which the mucosa generally maintains its normal color and appearance, the tube stops temporarily giving the sensation of elastic resistance that is most frequently observed at the level of the cricoid and diaphragmatic sphincters, although they may have another location.

The organic type, in which the tube stops giving the sensation of rigidity, of a different resistance, which creaks if you try to force it. In turn, the esophageal mucosa at the level of the stricture has lost its normal characteristics, appearing whitish or pearly in color, an indication of the presence of scar connective tissue. Inflammatory lesions may coexist. When faced with an organic stenosis, one always thinks of chronic inflammatory lesions or neoplasms.

Dilations

Characterized by an obvious increase in the esophageal lumen; They are commonly secondary to stenotic areas located below. Suprastenotic dilations are found in the esophageal axis itself and may correspond to lesions, both organic and functional. Organic dilations are of less importance, since the primary stenosing lesion that causes them is easily identified and they are never large.

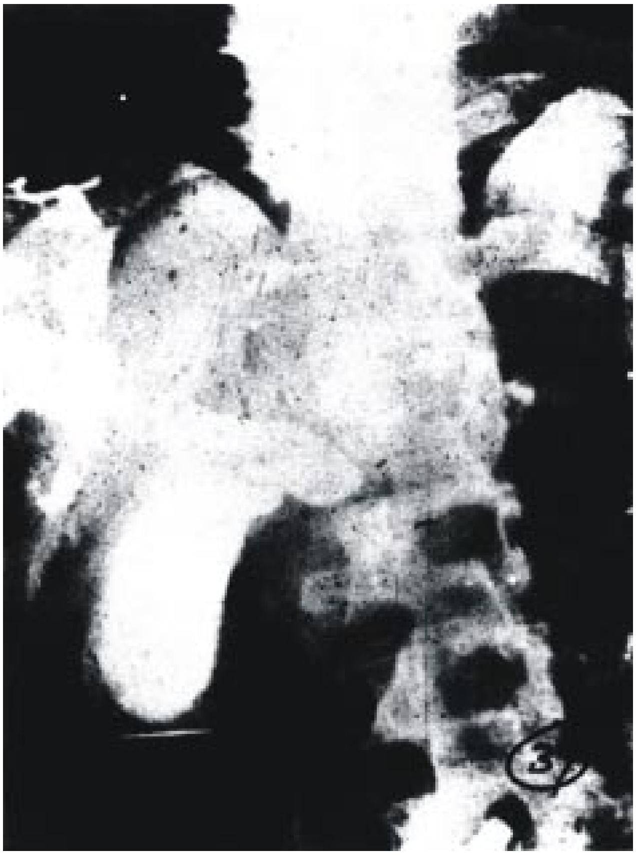
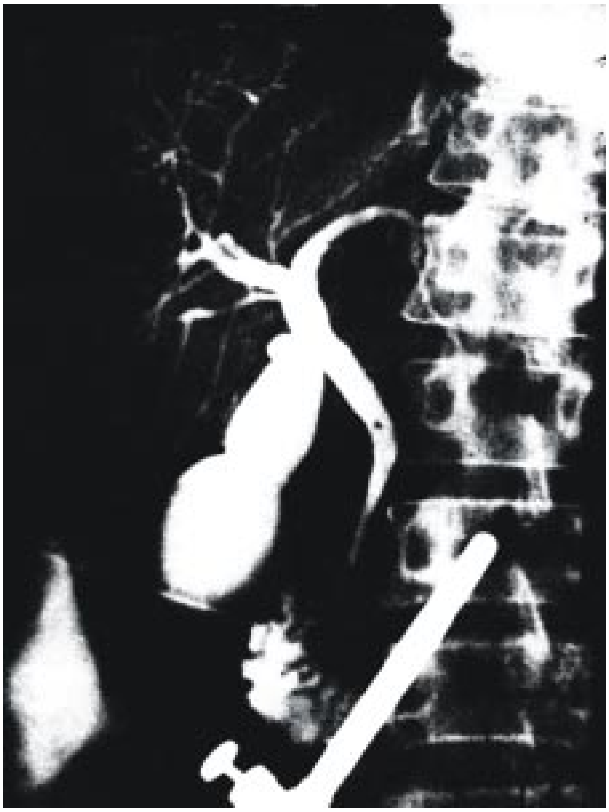


Fig. 62.12 Pancreatic neoplasia.

Fig. 62.13 Intrahepatic cholestasis (normal bile duct).



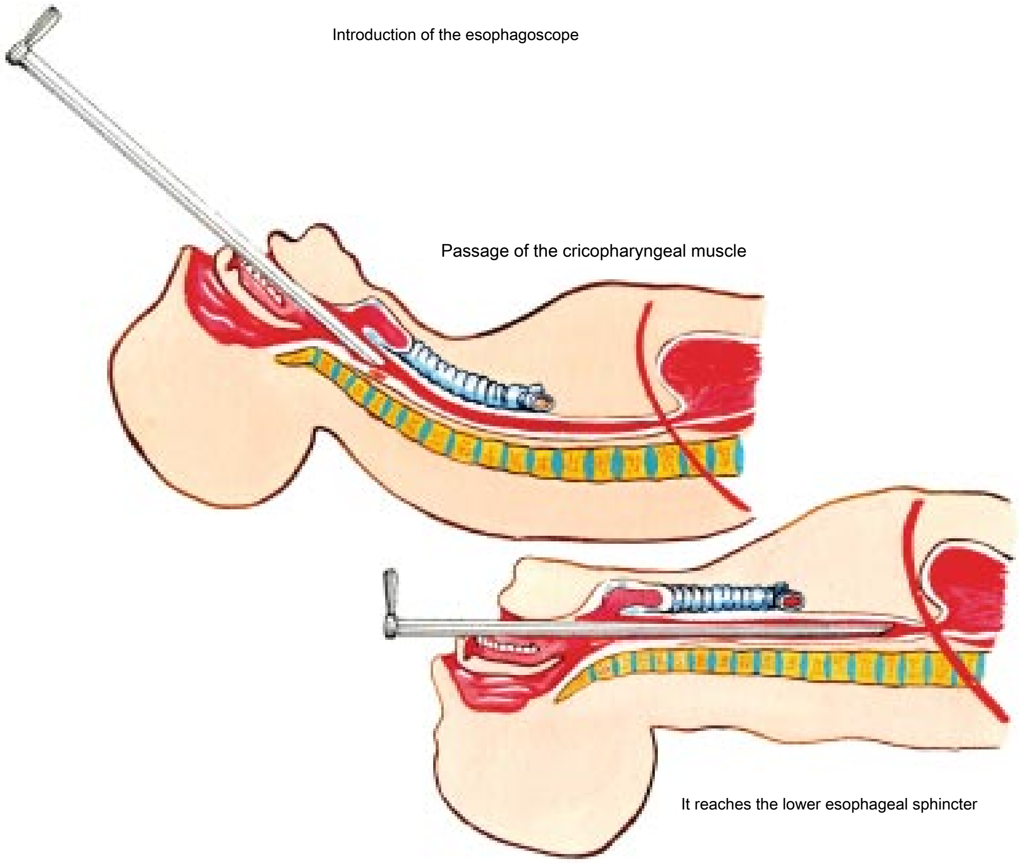

Introduction of the esophagoscope
Passage of the cricopharyngeal muscle
It reaches the lower esophageal sphincter

Fig. 62.14 Rigid esophagoscope and technique of its use.

Among the apparently functional dilations we must mention: achalasia. The mucosa may present inflammatory symptoms due to retention and the folds are thick. In general, it allows the passage of the esophagoscope to the stomach.

Parcel expansions

They correspond to anomalies and are located on the edge of the lumen: they are the dilations of the diverticula. At the entrance of these, a type of ridge can be seen that protrudes into the esophageal lumen, allowing the lumen of the diverticulum to be seen on one side and the esophageal lumen itself on the other. The mucosa that covers the diverticulum may or may not be altered. Three types of diverticula are basically found: the cricopharyngeal or Zenker diverticula, the thoracic or Rokitansky diverticula and the epiphrenic or Granet diverticula, the latter two being very rare.

Neoformations

Characterized by the presence of abnormal tissue in the esophageal

a) Proliferating, reminiscent of a cauliflower, in the form of bleeding mamelons upon contact with the equipment; other times ulcerated, irregular in appearance and with a tendency to narrow the lumen of the organ. We are referring mainly to malignant tumors of the esophagus, generally adenocarcinoma.

b) Better-looking, cleaner, sessile or pedunculated tumors, which correspond to benign tumors (adenoma, lipoma, fibroma, etc.).

The complementary biopsy is a necessity, since it confirms the differential diagnosis of benignity or malignancy and within malignancy, it details the histological type and the degree of activity, from all of which we deduce the therapeutic indications.

Areas of substance loss or ulcerations of the esophageal mucosa

They may constitute the primary element or coexist with other inflammatory or neoplastic alterations. In the first case, an isolated ulcer is seen, which presents with variable alterations in its bottom and regular or irregular borders, which also indicates benign or malignant ulcer. Let us take as an example the benign ulcer, which is found precisely above the cardia.

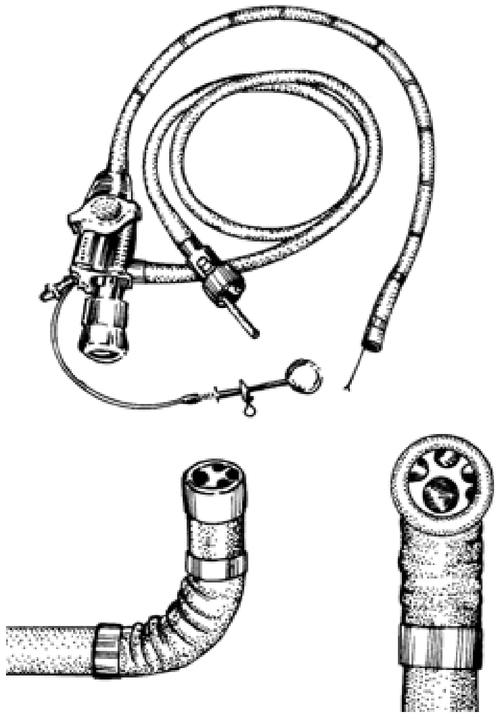


Fig. 62.15 Flexible esophagoscope: a, flexible biopsy forceps in place; b, close-up view of distal end, flexed; c, front view of the distal end showing one imaging beam (fiber), two light beams (fiber), and channel for the forceps.

Changes in color and appearance of the mucosa

They are generally diffuse, corresponding to inflammatory processes, from congestion to sepsis of the esophageal mucosa, known as esophagitis. Acute types are observed, although less frequently, generally determined by caustic substances and which can create exudative, phlegmonous suppurative or necrotic lesions.

Chronic esophagitis is more frequent and easier to verify with changes in the color and appearance of the mucosa, which appears red, with whitish or yellowish-white plaques, exudative in nature and others of a hemorrhagic nature, with more or less friability of the mucosa. Chronic esophagitis can coexist with strictures, as we noted, or with a hiatal hernia.

foreign bodies

They are easily diagnosed and can be removed, freeing the patient from surgery.

Vascular disorders

Finally, through esophagoscopy we can observe this type of alteration, especially esophageal varices, in cases of portal hypertension of different etiologies.

Complications

They are exceptional, although they can occur with the handling of rigid and open esophagoscopes in inexperienced hands.

Gastroscopy

Indications for diagnostic purposes

Medical gastroscopy is indicated for the diagnosis of those stomach processes that, despite the resources used, could not be specified, particularly in cases of doubtful radiological diagnosis. It should not be indicated routinely and, if possible, it should be preceded by radiological examination.

1. Persistent gastric syndromes, without diagnosis.
2. Chronic gastritis of any type.
3. Gastric ulcer and differential diagnosis between benign and malignant.
4. Therapeutic evolutionary control of in some diseases, such as gastric ulcer.
5. Differential diagnosis between carcinoma and lymphoma.
6. Benign or malignant tumors.
7. Gastric polyps.
8. Nutritional deficiency diseases.
9. Imprecise digestive clinical pictures.
10. Upper digestive bleeding.

Indications for therapeutic purposes

1. Endoscopic treatment of gastric hemorrhage.
2. Control of gastrectomized patients.
3. Polypectomy.
4. Percutaneous endoscopic gastrostomy.

Contraindications

1. Esophageal obstruction (organic or functional), which constitutes an absolute contraindication.
2. Acute corrosive gastritis.
3. Cachexia, heart failure, aneurysm, permanent dyspnea, psychosis, extreme sensitivity, etc.

Teams

Gastrofibroscopes are completely flexible devices with a diameter of about 12 mm, which transmit the image and light through thousands of glass fibers.

They have 1 or 2 channels that allow air and water to be insufflated and also aspirated, as well as to take samples for biopsy.

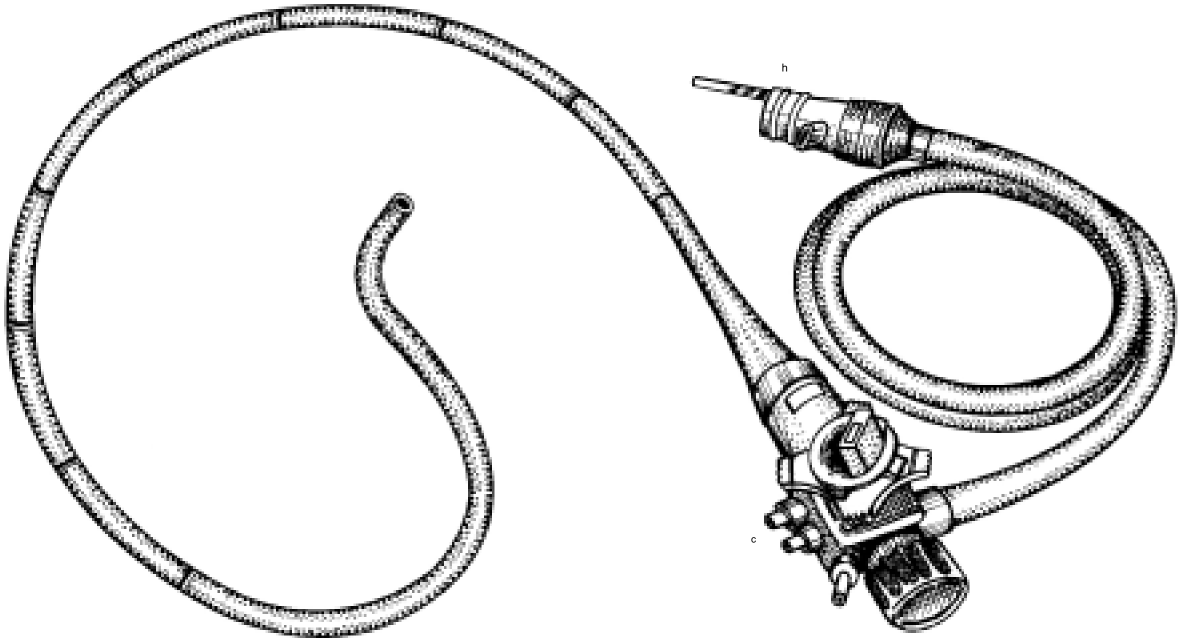
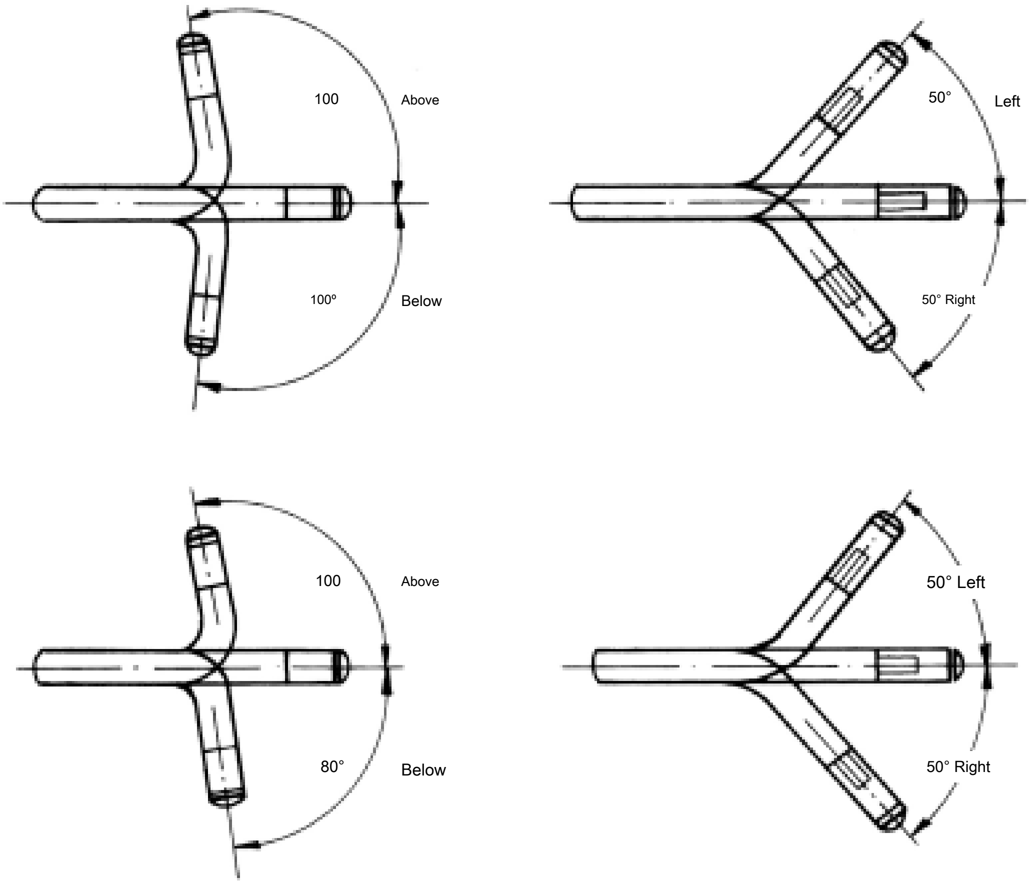


Fig. 62.16 Gastrofibroscope: a, cord to transmit light; b, connection; , controls section; d, tube being introduced; e, angular portion; f, final part.

Fig. 62.17 Positions of the distal portion of the fiberscopes.


100
Above
100º
Below
50°
Left
50° Right
100
Above
80°
Below
50° Left
50° Right

or for cytology (fig. 62.16). In turn, endoscopic surgery can be performed through these channels, removing polyps, coagulating bleeding, with lasers, etc.

The images can be photographed in color or transmitted by television or video for teaching. There are several pieces of equipment that differ in their caliber, length and angle of vision, which can be oblique or direct vision (gastroscope, duodenoscope, panendoscope, etc.).

An interesting feature that should be noted is the mobility (in all four planes of space) of the terminal portion of the equipment, which facilitates examination, including retrograde examination of the stomach (Fig. 62.17).

Technical procedure

After anesthesia of the pharynx, the introduction of the gastroscope is relatively simple and its entire passage through the esophagus can be easily controlled.

Once the instrument reaches the stomach (passing the cardia), air is insufflated to distend the gastric cavity and examine the entire mucosa.

Endoscopic guidance

A brief anatomical review of the stomach is essential, taking into account that gastroscopic anatomy is different in its terminology and details from the gastric anatomy that is studied to perform the physical examination (clinical anatomy) , as well as from radiological anatomy.

To learn to see in gastroscopy, endoscopic guidance is essential. It is necessary to imagine the stomach inflated and as if the subject were in the appropriate examination position of the left lateral decubitus.

We must distinguish the cardia, the pylorus, the lesser curvature, the greater curvature, the angle (point that separates the body from the antrum), the anterior wall, the posterior wall, the segments: body, antrum and fornix or highest portion.

It should be noted that, as a line of separation between the body and the antrum, there is a muscular fold that starts from the angulus (antri sphincter muscle). The cardia is delimited by the Z line that separates the esophageal mucosa (pale) from the orange gastric mucosa. The pylorus is not fixed, it varies from place to place in the endoscopic order and with the thickening of the muscularis mucosae.

We must know, as important information for gastroscopic orientation, that there are at least four morphological types of stomach (see Fig. 55.4), according to the angle formed by the upper two thirds more or less parallel to the axis of the body with the lower portions (antrum and pylorus). The most common types, however, are those in which this angle is 45 to 60 and 90º.

In these types, gastroscopic vision is relatively satisfactory and in those with a more acute angle (closed hook shape), with both portions almost parallel, vision becomes much less easy.

The abdominal wall comes into contact with the stomach to a good extent in the gastroscopic position, the antrum is supported on the pancreas.

Regarding the layers of the gastric wall, from a gastroscopic point of view, the serosa can be ignored; The muscularis mnucosae, except at the level of the pylorus, plays little role, the muscular layers only sometimes have physiological expression, the submucosa can sometimes appear due to transparency, but fundamentally we are interested in the mucosal layer that appears folded until we distend it with air.

In the physiological order, with the stomach full of air, the gastroscopist observes that peristalsis begins in the angle, oriented towards the antrum and the pylorus. Respiratory movements and arterial pulsations are also observable gastroscopically.

In Figure 62.18 you can see the correlation between the position of the gastroscope lens and the image that is observed, which helps to understand gastroscopic orientation.

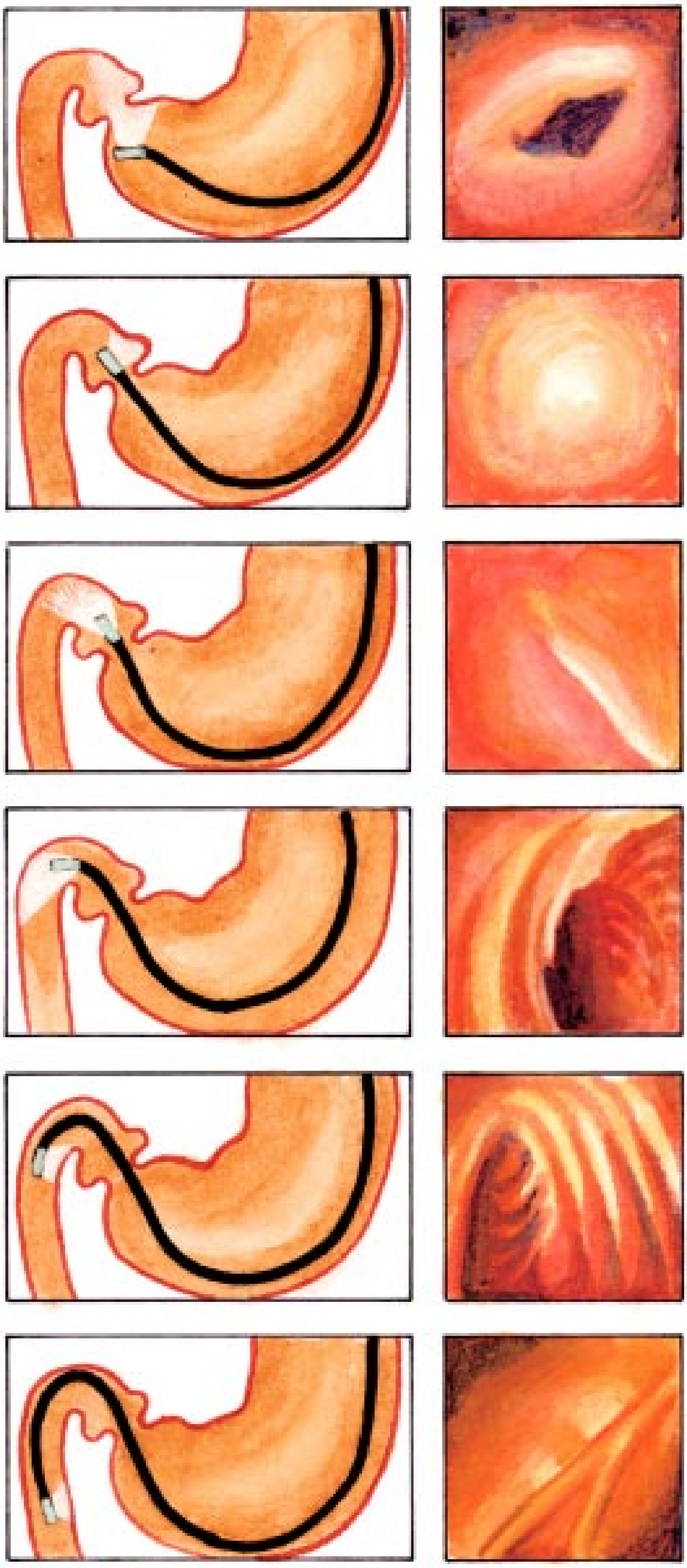
Normal data

1. The gastric mucosa has a smooth surface and is orange-red in color.
2. Respiratory movements are identified and vascular beats are also normally visible, sometimes with erethism.
3. When the gastroscope has just been introduced to any depth level, the mucosal folds appear immediately around the lens of the instrument. After inflation the folds are modified, being more marked in the greater curvature, as well as on the posterior face. In the lesser curvature and on the anterior surface, they are less numerous and less thick. They can disappear if the stomach is distended too much, which is not advisable as it is annoying and even dangerous. At the level of the antrum, the presence of folds is disputed, although sometimes they are perfectly visible.
4. Peristalsis is perfectly visible, sometimes some normal mucus is observed, and the pylorus is seen as a diaphragm or rosette, frequently confirming duodenal regurgitation.

Pathological data

Gastritis. It is the most common pathological alteration in gastroscopic examination. At the same time, the clinical and radiological diagnosis of gastritis is not always evident, which is why endoscopy is of great value.

Superficial gastritis are characterized by edema, congestion and more or less adherent secretion. The appearance of the mucosa varies from simple congestion to marked hypertrophy of the folds, sometimes with multiple exulcerations that can bleed, especially those caused by medications (Fig. 62.19).



Observation of the pylorus

Observation of the duodenal bulb

Observation of the bulbar recess

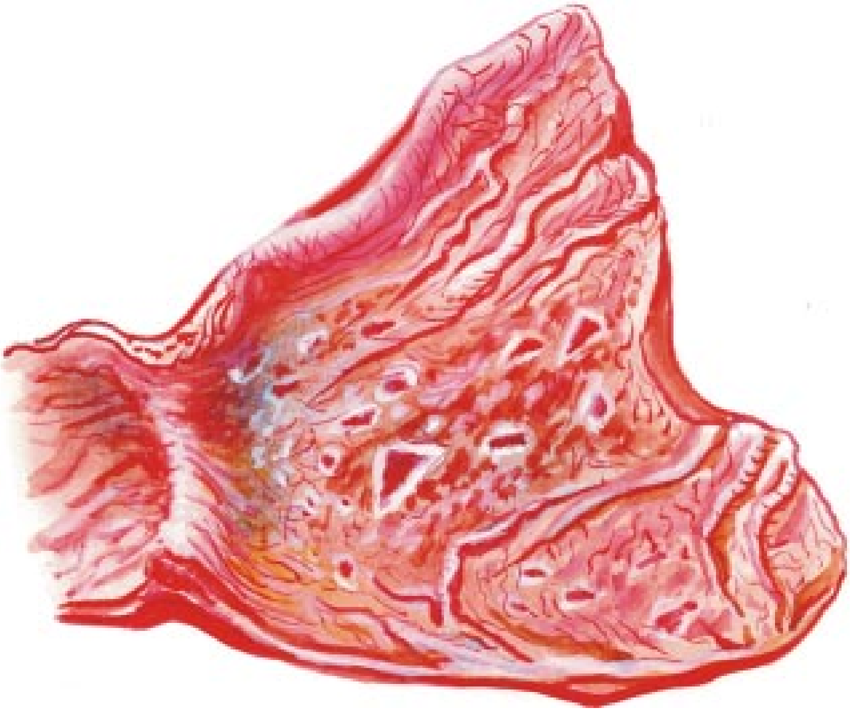
Postbulbar region and angle

Folds of the second duodenal portion

Observation of the amplicula of Vater

Fig. 62.18 Insertion of the gastroscope and regions observed.

Fig. 62.19 Superficial gastritis.



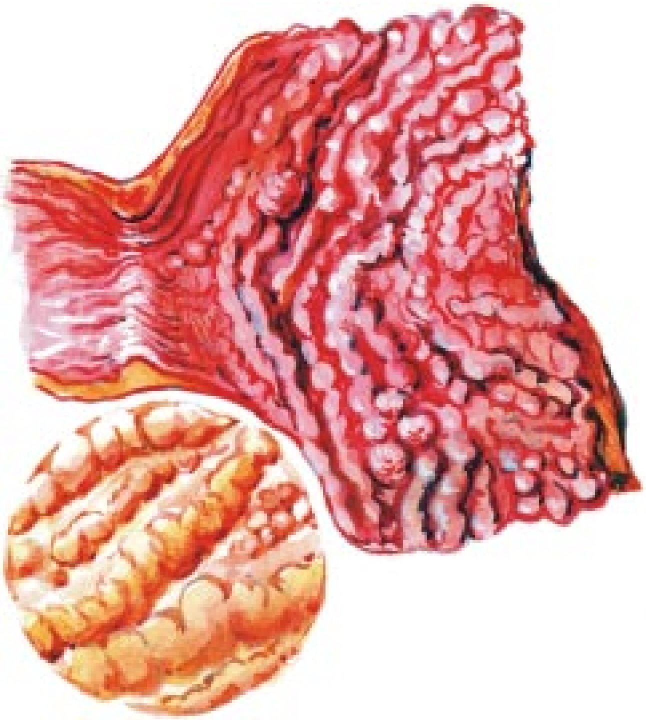
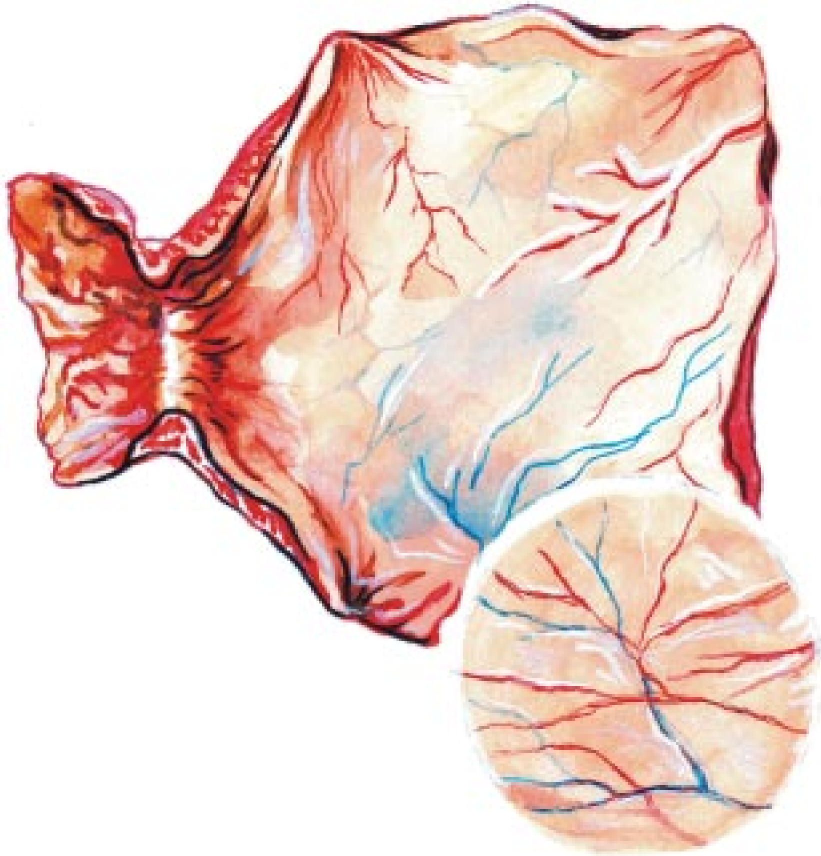


Fig. 62.20 Hepertrophic gastritis.

Fig. 62.21 Atrophic gastritis.



Chronic hypertrophic gastritis appear with typical gastroscopic images, characterized by a mamelonated appearance of the mucosa, more or less generalized or localized, which sometimes reminds us of the tumor appearance (Fig. 62.20).

Gastric atrophy or atrophic gastritis is another chronic process with an obvious endoscopic diagnosis. A thin, soft, pale pink mucosa is identified, which allows the vessels of the submucosa to be seen through transparency. The folds disappear completely or in areas (fig. 62.21). Gastric atrophy generally coincides with anachlorhydria, sometimes histamine-resistant, as occurs in pernicious anemia.

In summary, it is important to point out that through gastroscopy, superficial, hypertrophic and atrophic inflammatory processes or alterations can be identified in the same stomach in various combinations.

Gastric ulcer. As we know, this process is studied radiologically but gastroscopy can:

1. Confirm or deny a diagnosis.
2. Diagnose ulcers ignored in the radiological examination.
3. Distinguish the characters of benignity and malignancy.

   Any ulcer with an irregular base, a dirty bottom, torn edges, that gives the sensation of rigidity and infiltration and absence of peristalsis, is suspicious of malignancy.

   The opposite characters, of sharpness, clean background and regularity, will guide the benign. It is a diagnosis that requires gastroscopic experience and must be complemented with multiple biopsies of the border and fundus to definitively establish its etiology.
4. Assess the evolution of an ulcer, particularly its healing. On examination, it appears as a more or less deep loss of substance, with smooth edges and convergent folds that are arranged radially (fig. 62.22).

Benign tumors. They are generally identified as neoformations of the submucosa, which are observed as wide, soft elevations, with defined edges and a wide base and in which the mucosa that covers them is normal, they are sessile or pedunculated and the area surrounding the tumor is not modified. In any case, these characteristics are not absolute and when in doubt, only the biopsy will decide (fig. 62.23).

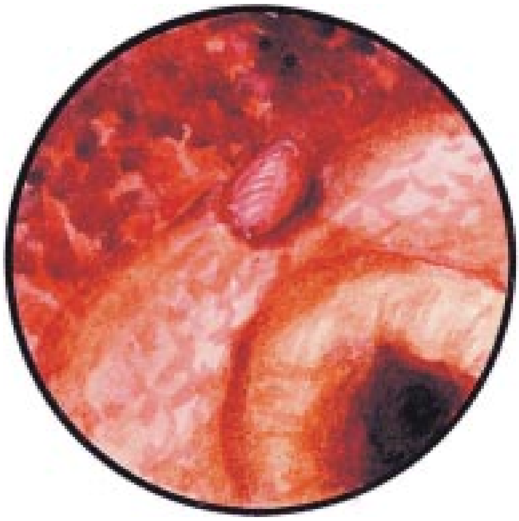


Fig. 62.22 Gastric ulcer located in the angular notch.

Malignant tumors (fig. 62.24). In most cases, a radiological diagnosis is made, as is also the case with benign tumors, but gastroscopy and biopsy allow a more precise decision on the malignancy of the lesion and the type of tumor. Neoplasms can take three macroscopic forms: the vegetative or proliferating form, which can present a polypoid, cauliflower, etc. appearance; the ulcerated form, which must be differentiated from the benign ulcer because it generally has more irregular indurated edges, with mamelons at the bottom and absence of peristalsis at that level, and the infiltrative form with more variegated characters and in which peristalsis disappears throughout the tumor area.

In incipient or early carcinomas, the Japanese classification is accepted, which specifies its most important characteristics. in detail but that go beyond the limits of a textbook. Sarcomas and lymphomas, which are much rarer, are also diagnosable. Lymphoma, in general, resembles the tumor types of hypertrophic gastritis.

Alterations of the operated stomach. This pathology, along with gastritis, is another success of gastroscopic diagnosis. The operated stomach, especially the gastrectomized one, offers the gastroscopist an easy view, since the residual stomach or stump is a regular cylinder that is easy to control endoscopically. On the other hand, radiology has obvious flaws in the operated stomach, especially when it comes to exploring the anastomotic mouth, a difficult site for barium to adhere and mold. For all these reasons, it is interesting to diagnose the

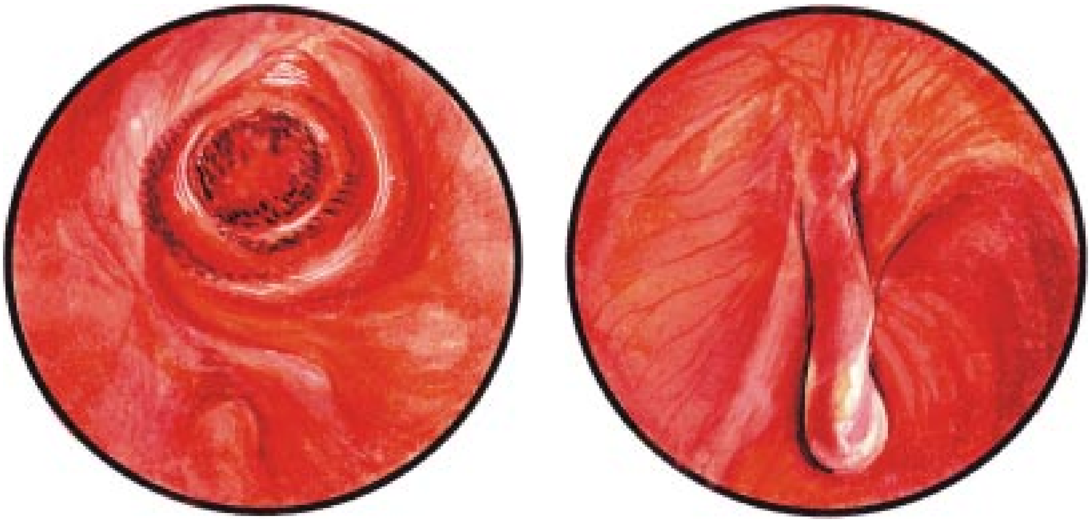


Fig.62.23 Benign tumors (polyps) of the stomach.

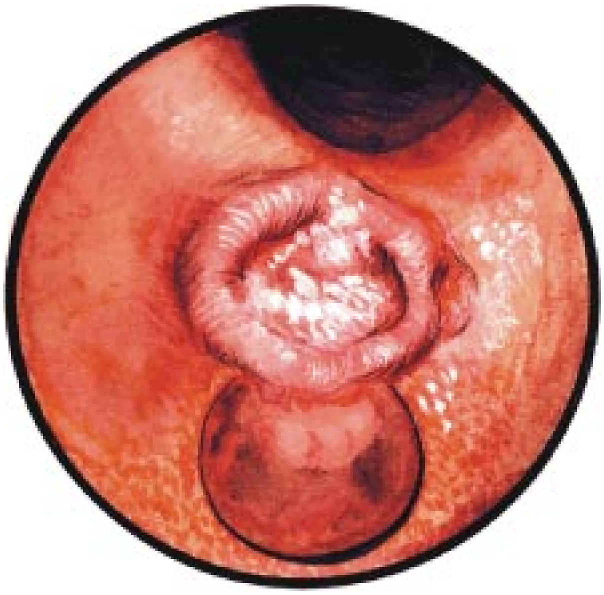


Fig. 62.24 Malignant tumor.

neomouth ulcers by gastroscopy, with which a bright and easy view of the lesion is obtained. Alterations of the mouth (stomatitis) are also identified, due to edema, deformity, suture threads, etc. The stump, also visible, allows the diagnosis of gastritis of the operated stomach and finally the anastomosed jejunal loop is identified, which allows the diagnosis of some jejunitis. Alterations of vagotomized stomachs (motor disorders, gastritis, etc.) are also identified. Carcinoma of the neomouth is extraordinary-

weird mind.

Rare alterations of another order. Through gastroscopy you can observe: syphilis, tuberculosis, other granulomas, fungal conditions, allergies, etc.

Alterations due to anomalies. Among these we have: diverticula, deformities, foreign bodies, etc.

In summary, gastroscopy is the most accurate method for diagnosing stomach diseases; cannot be surpassed by any other scanning method. It allows the precise diagnosis of gastritis, benign or malignant ulcers, neoplasms, etc.

It is the method of choice in upper digestive bleeding, of which it indicates the site, intensity and cause. In the operated stomach it defines the alteration causing the clinical symptoms.

At the same time, in all lesions it allows the evolutionary study and response to treatment, since it can be performed periodically given its simplicity and tolerance.

Duodenoscopy

Equipment

In this technique, the duodenofibroscope is used, a somewhat thinner and longer piece of equipment (Fig. 62.25) through which pathologies of this segment of the digestive tract can be diagnosed.

Indications for diagnostic purposes

1. Diagnosis and evolutionary assessment of duodenal ulcers, which reach a frequency of 70-80% of all ulcers (fig. 62.26).
2. Duodenitis, especially parasitic ones (giardia, strongyloid, necator), whether superficial or adopting the pseudopolypoid form, are easily diagnosed through complementary biopsy.
3. Diverticula and compression strictures (pancreas, gallbladder, etc.) may be required.
4. Diagnosis of upper digestive bleeding.
5. By visualizing the ampulla of Vater, tumors (ampullomas) or inflammations (odditis) in that area can be diagnosed.
6. The papilla can be catheterized and retrograde pancreatocholangiography performed, discussed later, which is of great value for the study of pathologies of these organs.

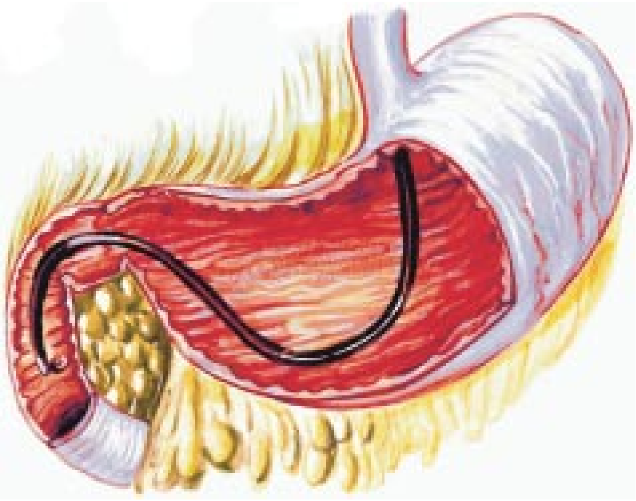
Indications for therapeutic purposes

1. Through endoscopic surgery, the sphincter of Oddi can be sectioned in the case of fibrous odditis, or residual stones can be removed from the common bile duct.
2. Endoscopic treatment of digestive bleeding

you go high

3. Polypectomy.

Fig.62.25 Duodenoscopy.



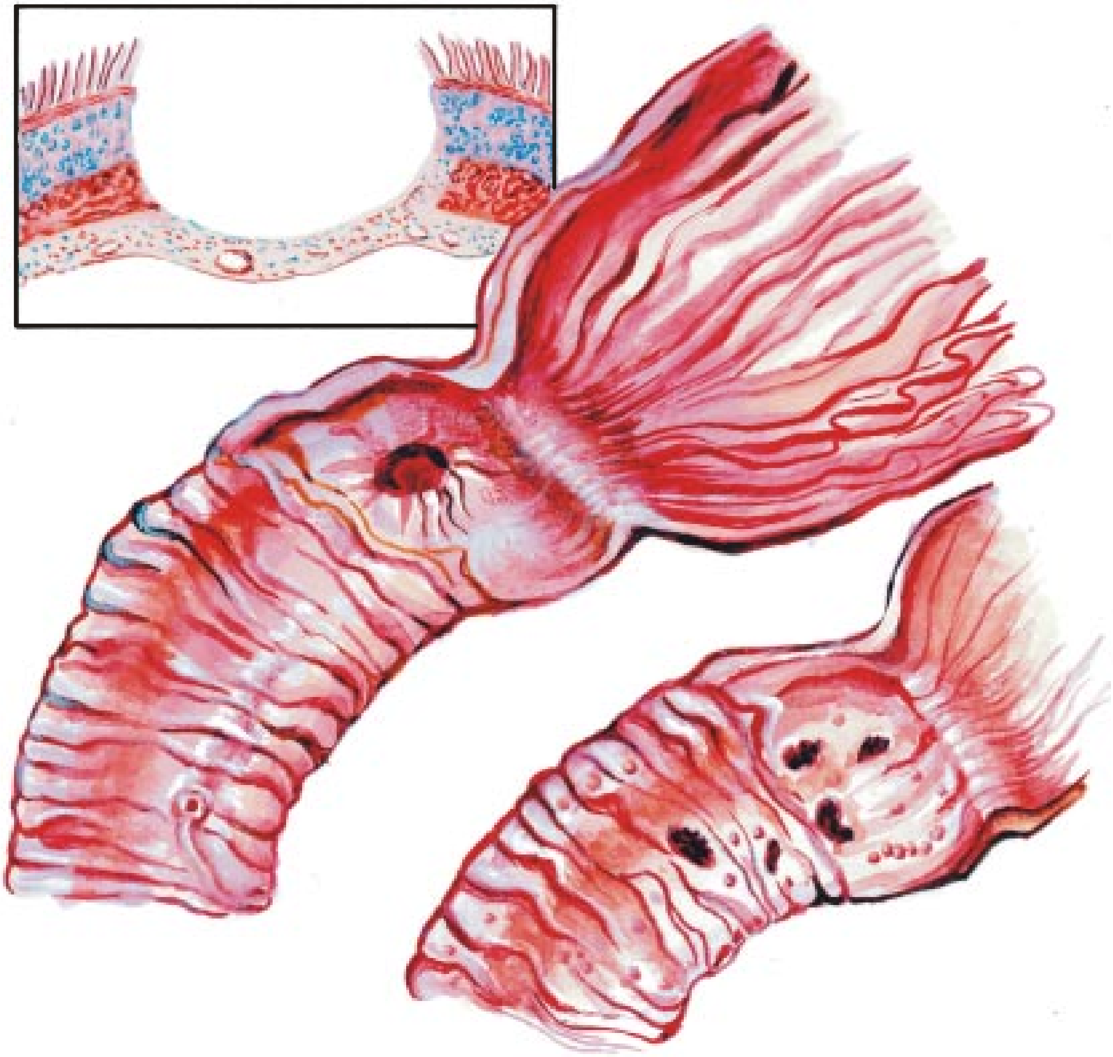


Fig. 62.26 Duodenal ulcer.

Panendoscopy

In this method, the panendoscope is used, whose view is frontal and which allows visualization of the esophagus, stomach and duodenum in the same examination.

It is one of the most used procedures, because it provides data on the three segments of the digestive tract, so its indications can be the same as those listed for each segment, although it reaches its maximum value in hematemesis, since it enables the topographic and etiological diagnosis of bleeding and its subsequent evolution. Additionally, the use of laser beams through the equipment can instantly control bleeding in many cases. It is also indicated in the study of anemic syndrome of unspecified etiology.

ENDOSCOPIC CHOLANGIOPANCREATOGRAPHY (EPC)

It constitutes an endoscopic-radiological procedure that consists of identifying the major duodenal papilla through an endoscopic examination, cannulating it and injecting iodinated contrast, thereby opacifying the bile ducts and pancreatic ducts. Its use has diagnostic and therapeutic purposes.

Indications for diagnostic purposes

1. Study of the jaundiced patient with suspected biliary obstruction.

2. Assess the patient without jaundice whose clinical picture suggests pancreatic or bile duct pathology.

3. Assess the possibility of pancreatic cancer when imaging studies are confusing or normal.
4. Assess chronic pancreatitis.
5. Preoperatively evaluate patients with pancreatic cancer or chronic pancreatitis.
6. Diagnosis of a pancreatic pseudocyst that has not been detected by imaging, and for pseudocysts known before planned surgery.
7. Study of the sphincter of Oddi and bile ducts using manometry.

Indications for therapeutic purposes

1. Residual or recurrent choledocholithiasis.
2. Papillary stenosis or sphincter of Oddi dysfunction.
3. Place a prosthesis (to drain the bile duct) or dilate a stricture.
4. Section the fibers of the minor sphincter of the ampulla.
5. Parasitosis (such as fascioliasis).
6. Biliary fistula.
7. Primary sclerosing cholangitis (to drain the bile duct using a prosthesis).
8. Choledochocele.
9. Acute biliary pancreatitis (for drainage purposes and to resolve the obstruction).
10. Acute obstructive suppurative cholangitis (also for drainage purposes and resolving obstruction).

To execute most of the therapeutic indications of EPC, it is necessary to perform an endoscopic sphincterotomy, which consists of sectioning, using electrocautery, the fibers of the sphincter of the papilla, with the aim of allowing the prosthesis to be placed to achieve drainage, remove the cause of the obstruction or dilate the stenosis.

ENDOSCOPIES OF THE RECTUM AND COLON

The most used methods are:

- Rectosigmoidoscopy.
- Colonoscopy.

Rectosigmoidoscopy

Concept

Rectosigmoidoscopy is a fundamental examination in the diagnosis of diseases of the rectum and sigmoid, and its objective is to visualize the mucosa of these segments by introducing equipment made for this purpose through the anus.

This examination is very simple and free of danger, as long as it is carried out by well-trained personnel. trado. It does not require anesthesia and does not cause great discomfort, except in those patients who have active anal lesions (fissures, ulcerations, hemorrhoids).

Colon diseases are located in the rectum and in the last portion of the sigmoid in 75% of cases, hence the great importance of this endoscopy and its usefulness in the diagnosis of this group of diseases. Endoscopy allows not only the observation of the mucosa, but also the taking of biopsies for an anatomopathological study.

Indications and contraindications

Rectosigmoidoscopy is indicated in any patient who manifests intestinal symptoms, that is, constipation, diarrhea, bleeding, presence of straining, phlegm, rectal pain, pruritus, etc., and in screening for polypoid and cancerous lesions.

There are practically no contraindications except for very acute lesions, which make the examination intolerable for the patient, and also certain stenoses when they are very intense, since there is a danger of perforation.

Teams

Rigid rectosigmoidoscope

It is a cylindrical metal tube 20-30 cm long and 20-25 mm in diameter, graduated on the outside in centimeters and has a mandrel stopper to facilitate its introduction. It also has a lighting accessory that can be internal (distal) or external (proximal) and a system of bulbs to insufflate air adaptable to a device that allows the entry and exit of air to be regulated at will by the endoscopist (fig. 62.27).

Flexible rectosigmoidoscope (rectosigmoid fiberscope)

It is a cylindrical tube made of plastic material, 60 cm long, completely flexible, whose distal end has four movements (up, down, right and left), forming an angle up and down of 170°, and to the sides of 140°. In the proximal portion it has controls, which allow the tip of the equipment to be directed. There are also two buttons at this upper end that make it possible to insufflate air, aspirate secretions and inject water to cleanse the mucosa of secretions (fig. 62.28).

These devices also have a special attachment to connect them to the light source, which is what provides illumination to the equipment, and another similar attachment to connect it to another source that allows polyps to be removed using electrocoagulation.

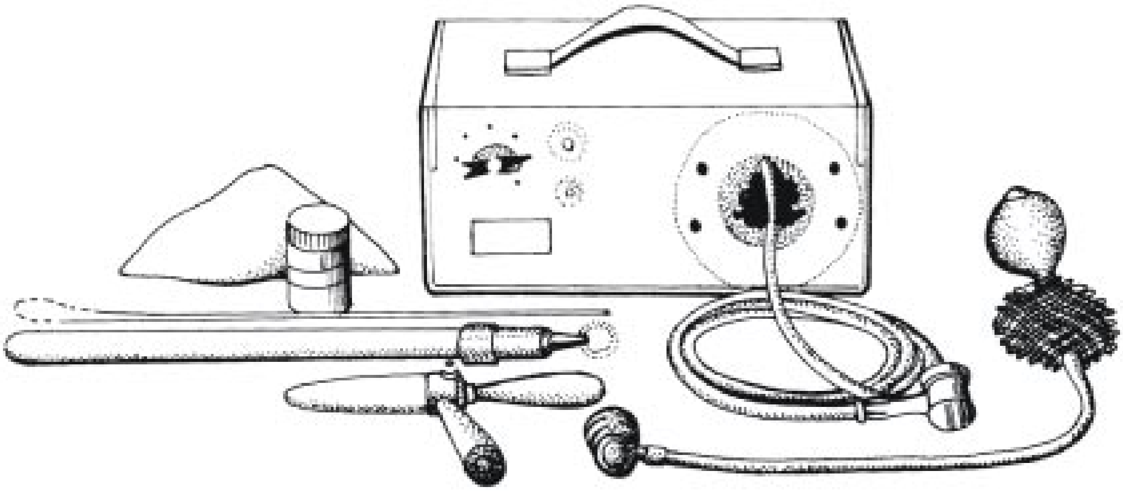


Fig. 62.27 Rigid rectosigmoidoscope.

The optical system can be focused according to the needs of the endoscopist.

Biopsy forceps, loops for resecting polyps and other instruments can be passed through a channel that runs the entire length of the equipment.

A camera is adapted to the eyepiece that allows the different endoscopic images to be photographed.

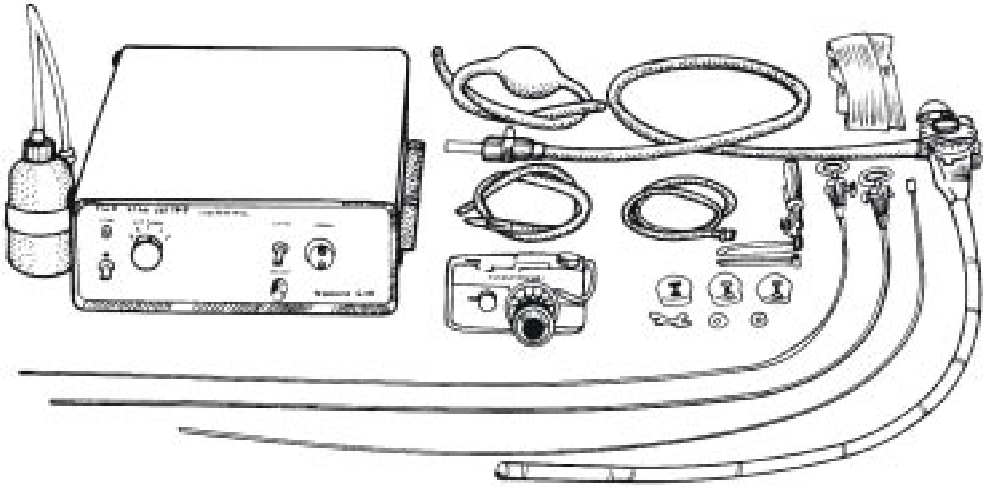
Semiotechnics

Cleaning the rectum and sigmoid is an essential requirement to ensure the success of the endoscopy, which will be done through previous enemas and a zero-residue diet.

Patient position for rectosigmoidoscopy (Fig. 62.29)

There are several positions, the most widespread is the inverted one, whereby the patient remains with his head down and his hips up, resting his elbows on a special support that the table created for this examination has for this purpose.

Fig. 62.28 Flexible rectosigmoidoscope.



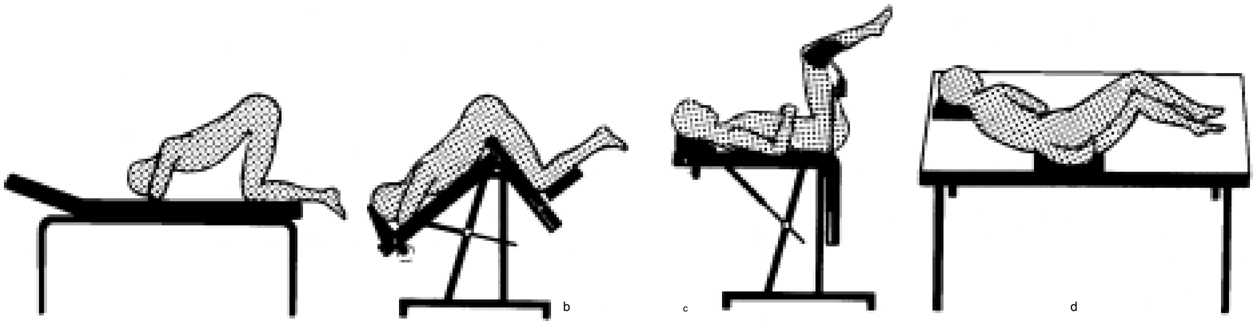


Fig. 62.29 Patient positions for rectosigmoidoscopy: a, genupectoral position on a horizontal rigid table; b, anterior position on proctological table; c, gynecological position; d, Sims position.

When this table is not available, the genupectoral position can be used and in some cases the Sims position, which consists of placing the patient in a left decubitus position, with the lower limbs flexed.

Equipment introduction

Rigid rectosigmoidoscope. The distal portion of the equipment is lubricated and it is introduced until it passes the internal sphincter. The mandrel is extracted and its progression is continued under vision. The introduction should be done gently, changing the direction in three directions: first towards the navel, then towards the sacrum and again towards the navel (fig. 62.30).

Flexible rectosigmoidoscope. As with the rigid one, the inspection and rectal examination are carried out previously. Subsequently, the equipment is lubricated with water-soluble lubricant, it is introduced under direct vision, and it will be advanced slowly as long as the light of the organ is visible.

By reaching the sigmoid-descending junction, one is sure to have explored the entire sigmoid.

Normal data

The mucosa of the rectum and sigmoid is smooth, moist in appearance and pink in color, a shade that varies from one patient to another. When the flexible equipment is used, which enlarges the image, the normal vascular network can be observed.

At the level of the anus, the columns of Morgagni, the semilunar valves and the hemorrhoidal plexuses are observed. Already in the rectum, the Houston valves are identified, which are generally three, although sometimes there may be four (see Fig. 16.13, volume 1).

These valves allow the rectum to be identified, which is also characterized by having a wider lumen than the sigmoid. In the sigmoid mucosa we observed numerous folds that allow us to identify the sigmoid.

Passing the rectosigmoid junction (13 cm from the anus) is sometimes difficult with rigid equipment, but generally Generally, with flexible equipment it is easy, and many times it is passed through it almost without the endoscopist noticing it, and without discomfort for the patient.

Pathological data

The most common lesions are tumors, inflammatory processes and hemorrhoids.

Let's briefly consider the first two.

Tumors

Among the tumors are adenomatous polyps, juvenile polyps, villous tumors, carcinomas and pseudopolyps.

Adenomatous polyps can be seen in different sizes, from 2-3 mm to exceptionally larger 2-3 cm. They may or may not have a pedicle, and their surface is smooth. As for color, they are generally redder than the mucosa on which they grow, this character being more prominent especially in the peduncles (fig. 62.31).

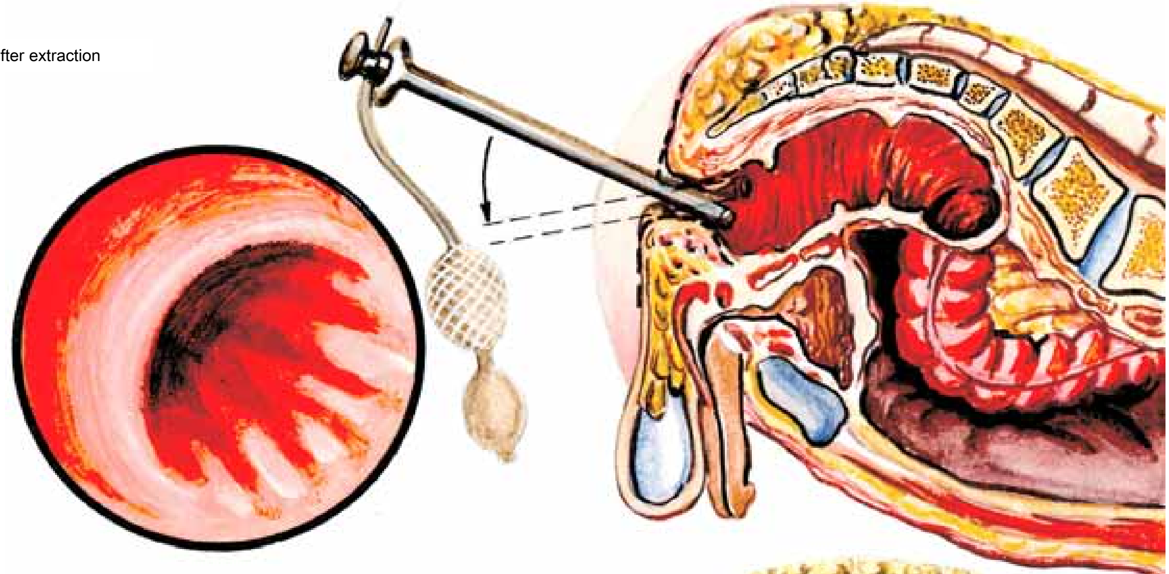
The juvenile polyp is usually very red. Villous tumors have a somewhat irregular surface and are very soft in consistency, which is why they sometimes escape rectal examination.

Carcinomas generally present as highly vascularized mamelonating masses, sometimes ulcerated, reminiscent of a cauliflower, this appearance corresponding to the vegetative form (fig. 62.32). The scirrhous form does not project towards the lumen, but rather infiltrates the wall, generally producing a stenosis.

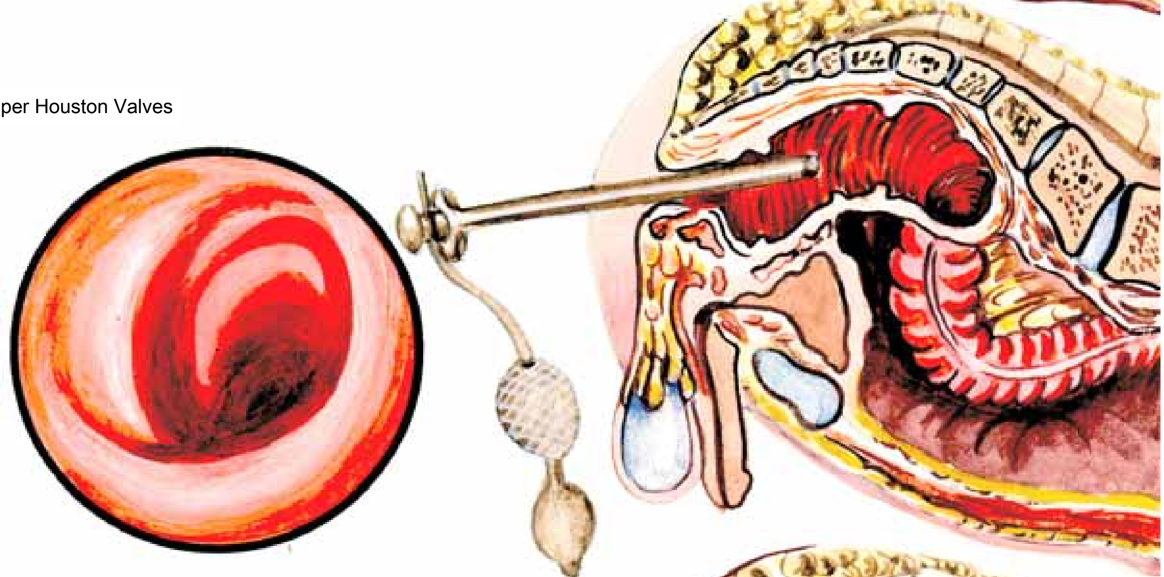
Inflammatory processes

Inflammatory processes can be acute or chronic and cause rectocolitis of different etiologies, which present endoscopically with changes in the color of the mucosa, hyperemic and hemorrhagic lesions, erosions, ulcerations, friability, abnormal secretions, exudates, false membranes and vegetations. All these findings vary depending on the etiological type.

Inferior rectum after extraction
the shutter



Middle and Upper Houston Valves



sigmoid colon

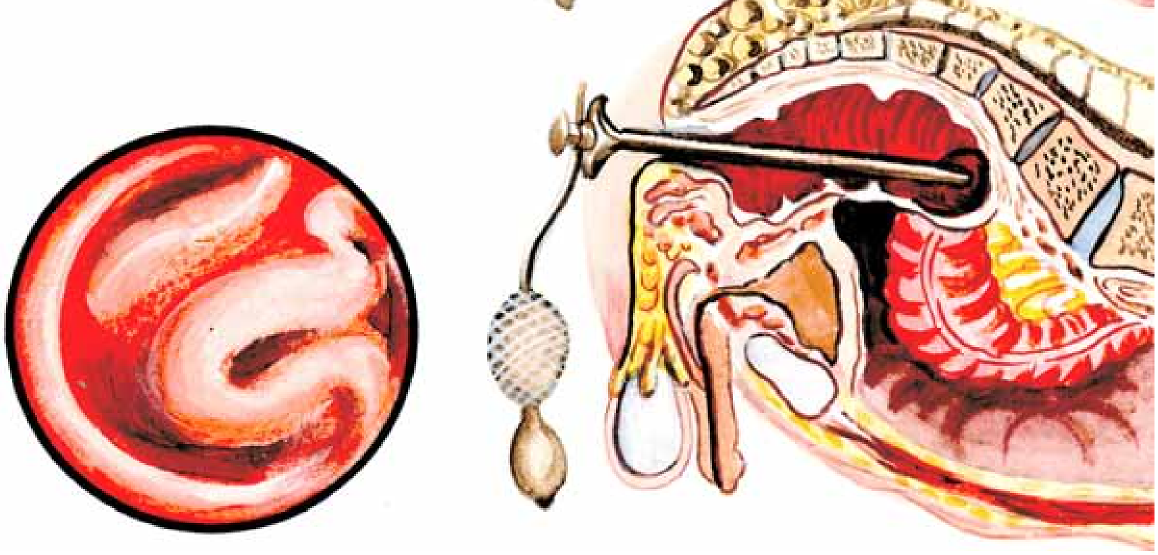
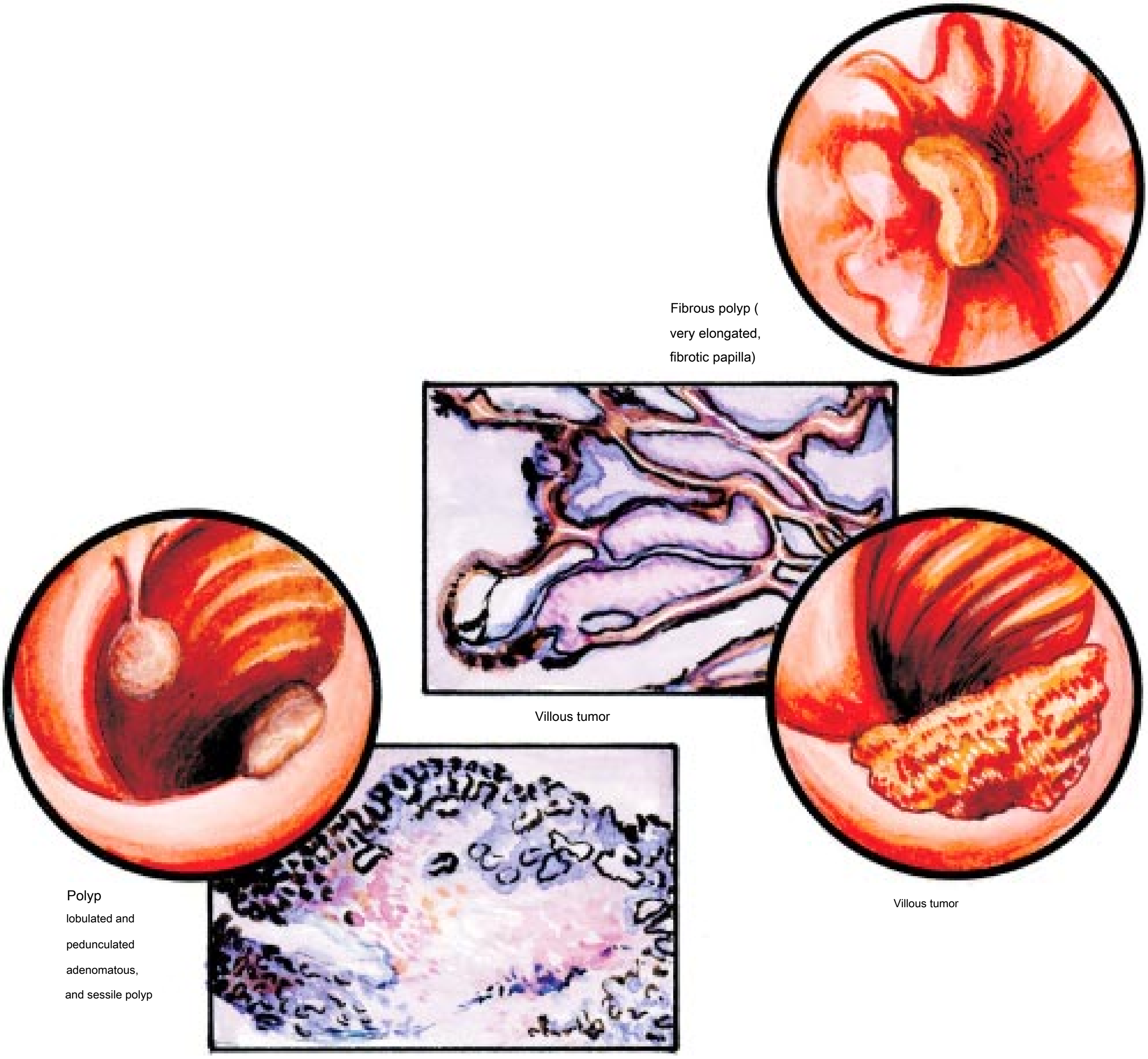


Fig. 62.30 Rectosigmoidoscopy technique with rigid equipment.



Fibrous polyp (
very elongated,
fibrotic papilla)

Villous tumor

Polyp
lobulated and
pedunculated
adenomatous,
and sessile polyp

Villous tumor

adenomatous polyp

Fig. 62.31 Polyps.

Among the inflammatory processes we mention:

Rectocolic amoebiasis with its typical image of oval-shaped ulcers, with sharp edges and a bottom covered with mucopus, separated from each other by healthy mucosa (fig. 62.33).

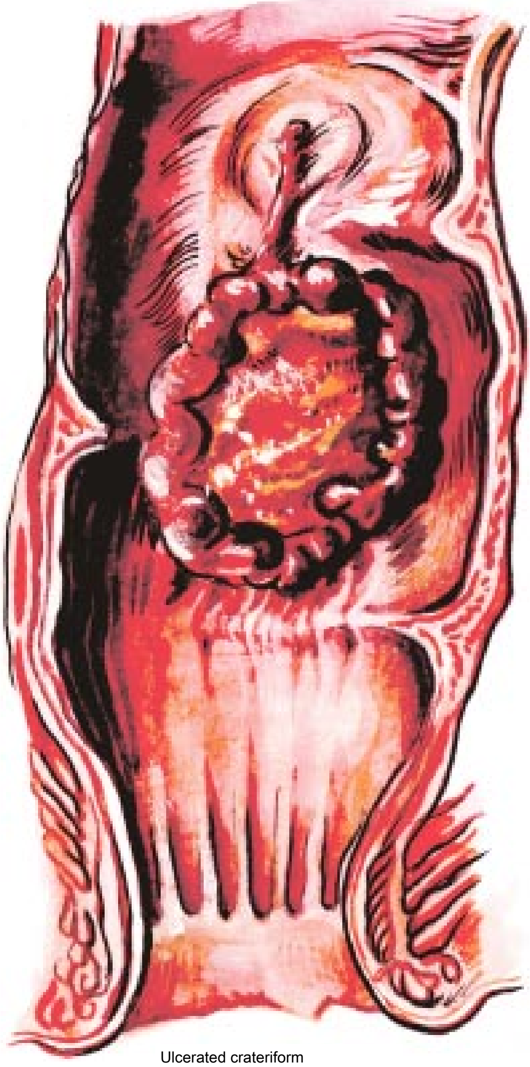
Bacillary dysentery, where the inflammatory process affects the entire mucosa, appearing hyperemic, easily bleeding, with superficial ulcerations that are smaller than those observed in amoebiasis, and sometimes covered by an adherent membrane of the diphtheroid type (fig. 62.34).

Idiopathic ulcerative colitis, which appears with its typical image of infiltration, friability, erosions, bleeding, purulent secretion and exulcerations or loss of substance in its acute phase and the typical image of granular, "sandpaper" mucosa, in the chronic phase, which in the terminal phase is expressed by the mamelonated or polypoid appearance, with strictures.

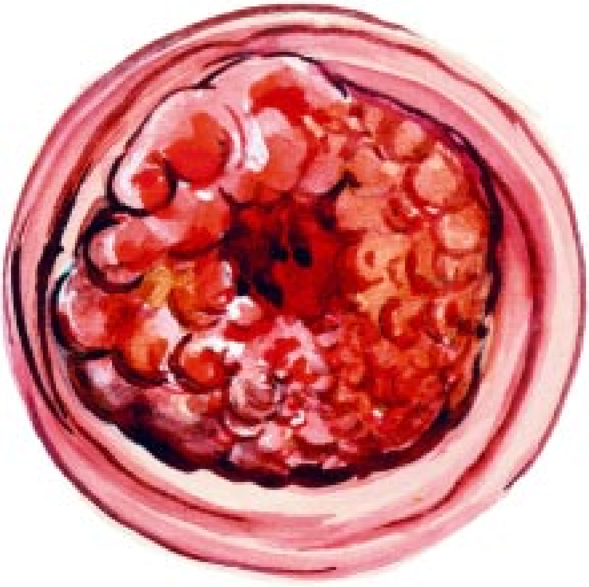
Colonoscopy

Concept

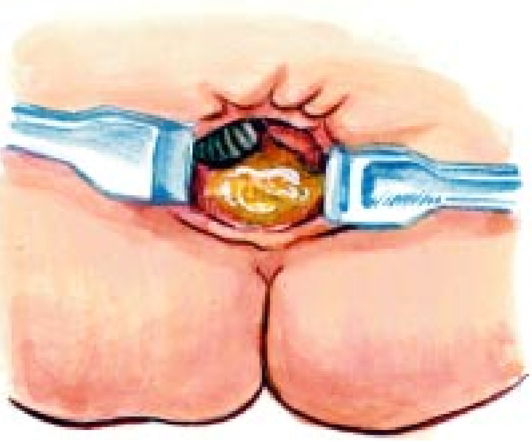
Colonoscopy is an examination that consists of viewing the entire colon through the introduction of flexible equipment made for this purpose: the colonoscope.



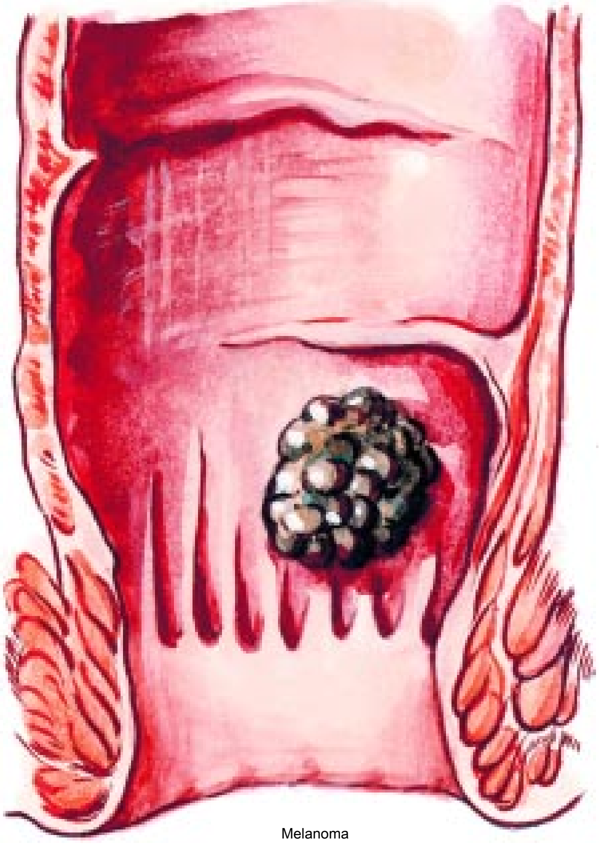
Ulcerated crateriform adenocarcinoma of the upper



Adenocarcinoma completely surrounding the lower rectum (sigmoidoscopic view)

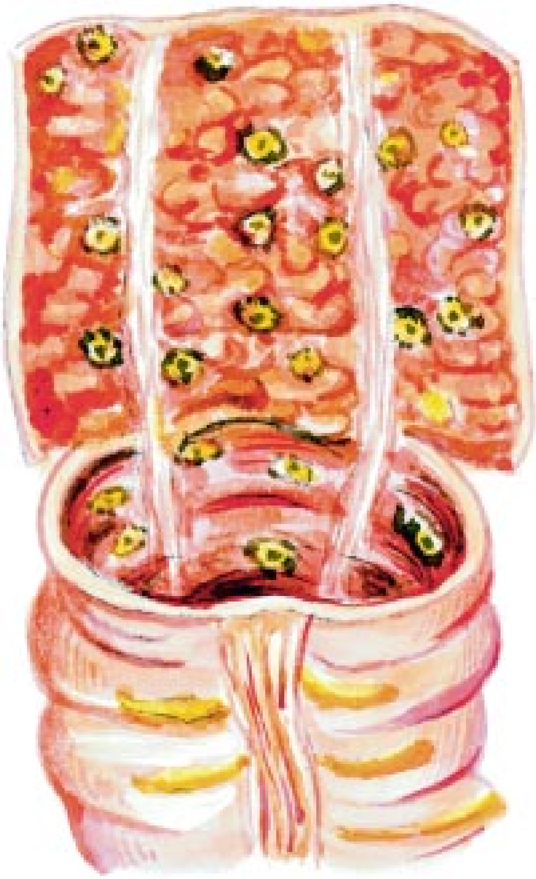


Anal canal epithelioma

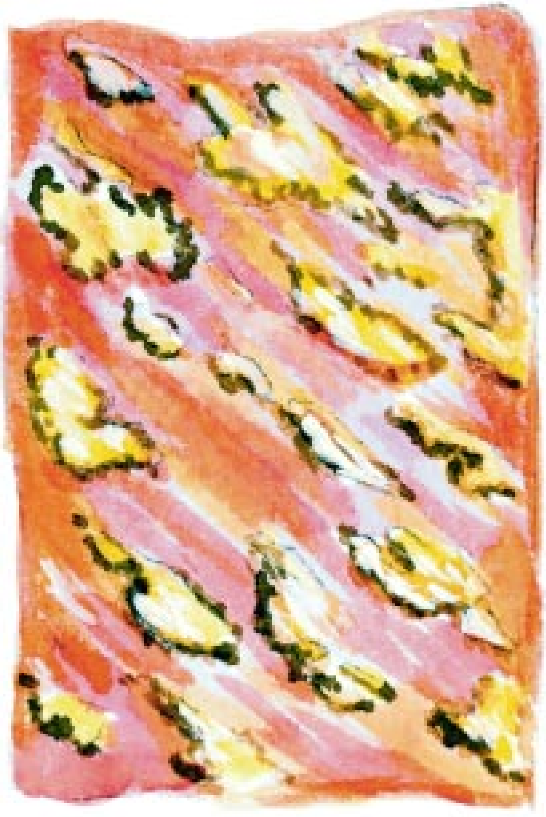


Melanoma

Fig. 62.32 Malignant tumors of the rectum.



Large intestine segment: amoebic colitis of moderate degree, small raised ulcers, and mined edges



Severe amoebic colitis, now rarely observed; large areas with ulcerations due to the confluence of small ulcers. Ulcers have torn the undermined edges; congested mucosa intervenes.

Fig. 62.33 Amebiasis.

The colonoscopic examination is primarily used to evaluate the possibility of cancer, inflammation, and bleeding. Its indications can be for diagnostic and therapeutic purposes.

Indications for diagnostic purposes

1. Rectal bleeding, mucus, etc., with normal results in rectosigmoidoscopy.
2. Patients with indicative symptoms and with radiological evidence or suspicion of colonic disease.
3. Diagnosis of inflammatory diseases of the colon, mainly to obtain biopsies, know the extent of the process and for its evolutionary control.
4. Diarrheal symptoms of unspecified etiology.
5. Patient with chronic iron deficiency anemia of undetermined cause.
6. Evolutionary control after endoscopic polypectomy or surgical resection.
7. Detection of polypoid lesions and colon cancer in at-risk populations (genetic predisposition and others).

Indications for therapeutic purposes

1. Endoscopic polypectomy.
2. Dilation of postsurgical and inflammatory strictures.
3. Revision of anastomosis, after tumor resection.
4. Treat hemorrhage from lesions resulting from vascular anomalies, ulcerations, etc.

Contraindications

1. Active anal lesions.
2. Fulminant colitis and colitis in the acute phase.
3. Multiple and extensive diverticulosis with large diverticula, due to possible contusion of the mouth of the diverticula with the lumen of the colon.

There are cases of relative contraindications such as heart and lung failure.

In some isolated cases, the endoscopist may be forced to suspend the examination due to lack of cooperation or poor preparation in colon cleansing.

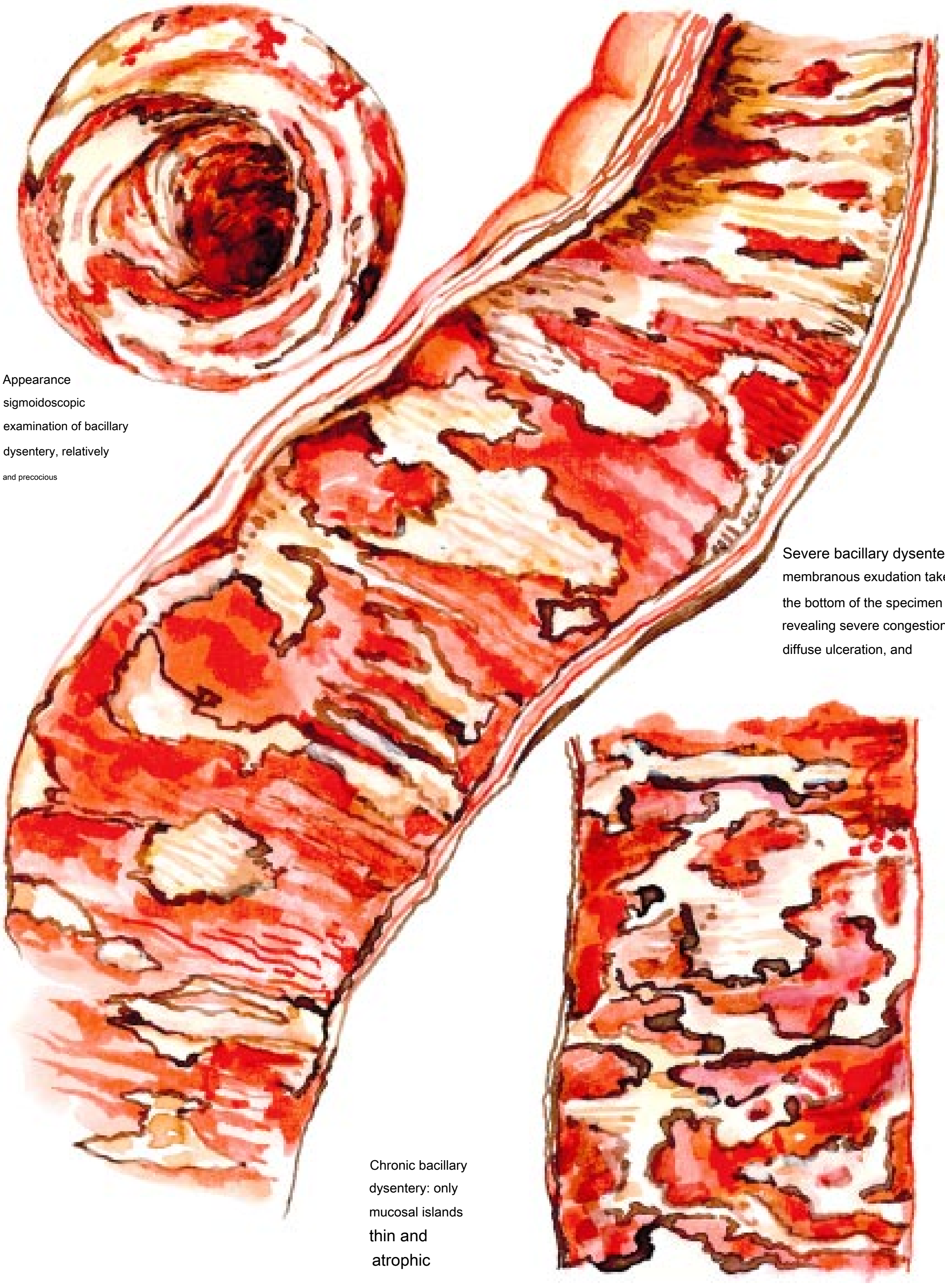

Appearance
sigmoidoscopic
examination of bacillary
dysentery, relatively
and precocious
Severe bacillary dysentery:
membranous exudation taken from
the bottom of the specimen
revealing severe congestion,
diffuse ulceration, and
Chronic bacillary
dysentery: only
mucosal islands
thin and
atrophic

Fig. 62.34 Bacillary dysentery.

Teams

There are several models of colonoscopes, which differ fundamentally in their length. The characteristics are the same as the sigmoidofibroscope but with different lengths. The short type is about 90 cm in length and the long colonoscope is 170 cm.

With current equipment, visual examination of the mucosa of the entire colon is achieved, from the anus to the cecum and sometimes, also the terminal ileum.

Semiotechnics

Patient preparation

This technique requires correct preparation of the colon, preferably antegrade, in order to achieve the maximum possible cleansing of the organ.

Patient position for colonoscopy

Of the positions indicated, the most used are the supine position and the Sims position.

Equipment introduction

After inspection and rectal examination, to detect any underlying pathology, a colonoscopy is performed.

The instrument is always introduced under visual endoscopic control, the intestinal lumen must be visible. If this is not the case, as may occur in a marked sinuosity of the sigmoid colon, the instrument can be carefully pushed forward while the mucosa can be clearly seen sliding in front of the tip.

It is not advisable to advance the instrument under radiological control, due to excess radiation for the patient and the equipment, which affects the glass fibers; However, occasional fluoroscopic control of the tip situation may be considered useful.

Air insufflation to open the intestine should be minimal at this stage.

The sigmoid colon can be passed relatively easily when it describes a simple curve; There are anatomical variations that can make the passage of the instrument very difficult, which is why an experienced endoscopist is required to perform it.

When the descending colon has been reached, the most difficult step is to overcome the splenic flexure, since it sometimes forms very narrow angulations that prevent easy passage of the equipment. Once this difficulty is overcome, we reach the transverse colon, which is easily identified by the triangular shape that the folds adopt at this level.

Once the transverse colon has been passed, another difficult moment is to transpose the hepatic flexure of the colon, which is also overcome with different maneuvers and sometimes even visualization under a screen is necessary.

Finally, once the right or ascending colon is reached, the passage of the equipment becomes easier, the cecum can be reached and the ileocecal valve and the appendicular orifice can be identified.

The data offered by colonoscopy are the same as those indicated in rectosigmoidofibroscopy.

EXPLORATORY METHODS BY PROBING

MEASUREMENT OF GASTRIC ACIDITY (GASTROKYMOGRAM)

It is a simple technique that does not require sophisticated or expensive material, although nasogastric intubation of the patient and fasting for 12 hours before the test is essential.

Initially, basal acid production is determined.

It has been demonstrated (Kay, 1953) that the gastric secretory response increases as the dose of secretion stimulants increases, until reaching a figure above which the responses are equal. Therefore, these doses represent a maximum and uniform stimulus of the HCl-secreting parietal cell. The most commonly used substance is pentagastrin, a synthetic gastrin peptide. The dose to obtain the maximum stimulus (6 ugkg) is injected intramuscularly and the maximum acid production will be obtained. More representative of the maximum acid secretion capacity is the so-called acid secretion peak, which is given by the two consecutive periods of 15 min each, or the three of 10 min, in which the highest acid output has been obtained.

Indications

Knowledge of gastric acidity has lost importance in a series of diseases in which years ago it was considered almost essential. Currently its indications are limited, but sometimes it is of great help in the diagnosis of certain gastric pathological conditions. In the study of a patient with hypergastrinenmia, measurement of gastric acidity is considered necessary to complement the diagnostic interpretation and define whether it is hypergastrinemia with hypoacidity or hyperacidity.

Contraindications

Contraindications are:

1. Esophageal obstruction (organic or functional).
2. Extrinsic compressions due to tumors.
3. Esophageal varices and acute corrosive gastritis.
4. Cachexia, heart failure, aneurysms, permanent dyspnea, psychosis and extreme senility.

Normal data

From a practical point of view we can consider that gastric secretion is an intermittent phenomenon and that two phases are distinguished:

1. Interdigestive phase (fasting). Also called fasting, basal secretion or nocturnal secretion; In it, the contents of the stomach are studied on an empty stomach, noting its volume, acidity, food remains, etc.
   This secretion would be directly related to vagal tone.

2. Digestive cycle (activity). After eating the test breakfast, the digestive cycle begins, in which three phases can be distinguished, which are: cephalic, gastric and intestinal, and which were already described in Chapter 55; However, from the point of view of the gastrochymogram we must point out some aspects, which we refer to below.

The cephalic phase is equivalent to the time during which contact of the food mass (test breakfast) with the gastric mucosa has not yet been established. This phase begins when the subject's sensory contact with the food is established (smell, vision of the food, thinking about it, etc.); Like fasting, it is related to vagal tone and secretion is established by a reflex mechanism, through vagal fibers and cortical and subcortical brain centers.

The gastric phase begins approximately half an hour after ingesting the test breakfast, extends up to 2.5 hours, and its increasing character reaches a maximum at 60-90 minutes. This secretion is established by the contact of the test meal with the gastric mucosa, at the expense of different factors that seem to influence it, studied in Chapter 55.

The intestinal phase begins at 2 hours and lasts for half to three-quarters of an hour. The secretion it produces is established and maintained under the stimulus of a substance (gastrin) that would be generated in the duodenum when it receives the gastric contents.

The normal values that are collected in the gastrochymogram are:

In the interdigestive phase (on an empty stomach)

1. The volume aspirated on an empty stomach should not be more than 50 mL.
2. Its color is grayish white.
3. Its appearance is transparent, fluid or somewhat viscous, depending on the amount of mucus.
4. Its smell is characteristic, penetrating and easily identifiable.
5. Normally it should not contain bile or only very slight traces of bile reflux.
6. Sometimes it is possible to observe few, fine filaments of fresh, red blood, of a traumatic type.
7. Contains no sediment or food debris.
8. Free and total hydrochloric acidity should not be higher than 25 and 40 mEq/L, respectively.
9. The pH is 1.5-2.
10. The difference between free and total acidity should be 5-15 mEq/L.

In the digestive cycle

The same characteristics should be noted as for fasting gastric juice, except that its color turns blue, due to the methylene blue used in the test breakfast, whose color in turn should become lighter until it disappears in the last sample or samples after 2 hours after starting the test.

It should be noted as a final point that if the chyme is intense blue and the final volume greater than 50 mL there will be a delay in evacuation; On the contrary, if the chyme is clear and the final volume is less than 50 mL, the evacuation is considered good.

Pathological data

Secretory alterations of the stomach must be reconsidered according to the modern data of this exploration.

They can be considered:

1. Default Alterations (Sims Ranking):
   a) True aquilias (without hydrochloric acid or enzymes), have pH 7.5-8.7, no response or no response after stimulation by histamine.

   b) Achlorhydria with a pH greater than 6, after histamine, without free acidity and without pepsin.
   c) Achlorhydria with a pH greater than 4.5 without proteolytic activity.
   d) Achlorhydria with proteolytic activity and pH above 3.5.

   e) The most frequent causes of all these alterations are gastritis (especially atrophic gastritis), cancer and an operated stomach.
2. Alterations due to excess:
   a) Hyperchlorhydria. They must be understood in the clinical sense rather than in the physicochemical sense, since the secretion by the parietal cells is in a constant concentration and the acidity cannot exceed 160 mEq/L.

It is seen in some gastritis and duodenitis but, above all, in duodenal ulcers and some gastric ulcers.

b) Gastric hypersecretion. The old terms of "gastrosurrhea", Reichmann syndrome, have not prevailed. It seems that these syndromes are ulcers of the duodenum or pylorus. The problem has increased with the description of Zollinger-Ellison syndrome, characterized by gastric hypersecretion with a considerable increase in output, increased acidity, presence of an atypical ulcer that sits on the distal duodenum or jejunum, and a non-insulin secreting pancreatic tumor. In addition to its clinical interest, this syndrome raises the problem of an active pancreatic intrinsic factor (gastrin) on gastric secretion. Hypersecretion persists after vagotomy and antrectomy. The gastric secretion figures can be considerable: 2-3 L of nocturnal volume. 60-110 mEq/L of free acid with a discharge of 600 mEq in 24 h. Any "out of bounds" gastric hypersecretion should suggest Zollinger-Ellison syndrome.

3. Other disturbances of gastric secretion:

a) Pepsin secretion increases in duodenal ulcer and decreases in cancer, being absent in pernicious anemia or Biermer's disease.

b) The secretion of glandular mucoproteins is increased in duodenal ulcers and hypertrophic gastritis. It decreases in cancers and atrophic gastritis and is absent in Biermer' s disease. Mucoproteases are increased in achlorhydric gastritis, in alcoholic gastritis and after gastrectomy. Its concentration is in inverse proportion to that of glandular mucoproteins.

c) A. Cornet has demonstrated, with histochemistry and histotorradiography methods performed with S35 sulfate, the alteration of mucus secretion in cases of intestinal metaplasia of gastric cells.

Kay test

It uses maximum stimulation with histamine according to weight, and consists of performing a constant aspiration of gastric juice, measuring the different components in total, on an empty stomach and every 15 minutes for 1 hour.

It allows the basal secretion and maximum secretion to be assessed by relating their absolute and relative values, which facilitates the approximate calculation of the so-called parietal component, that is, the mass of parietal or oxyntic cells.

Hollander test

Stimulation with insulin is carried out, causing hypoglycemia and in this way, through a vagal mechanism, the secretion of juice and hydrochloric acid occurs. This test is useful to study the vagal component before and after surgical vagotomy.

BILIARY DRAINAGE OR MELTZER-LYON TEST

It is a very useful study method within the limits and indications that we will point out, which should not be ignored in a qualified gastroenterology service. Let us also say that it is not a method available to everyone, but to be useful, it requires prepared and well-organized personnel.

Indications

Currently, the indications for diagnostic biliary drainage have been considerably limited: the most important are:

1. Non-calculous cholecystopathies, particularly dyskinesias.
2. Alleged parasitosis of the bile ducts.
3. Sequelae of cholecystectomies.
4. Some jaundice.
5. Suspected cases of gallstones and apparent loss of gallbladder function, in which the previous radiological examination could not be convincing.

Contraindications

They are the same as for gastric catheterization, that is, cases in which the tube should not be passed. Furthermore, it is important to remember that in gallstones due to small migratory stones, biliary drainage is not recommended, as we can cause the stones to be dislodged from the gallbladder and pass into the bile ducts, especially the common bile duct.

Normal data (rig. 62.35)

Bile A. It is obtained in an amount of 30-40 mL; It is lemon yellow in color, of low viscosity, with few flocs, and its output form is continuous; It comes from the thick bile canals and generally mixes with the gastric contents that pass into the duodenum, which produces turbidity of the bile, which is due to the precipitation of bile salts by hydrochloric acid. Microscopic examination reveals some leukocytes, mucus, duodenal epithelium and bacteria.

Bile B. It is obtained in an amount of 50-60 mm; It is mahogany brown, blonde fish or havana tobacco, transparent, with a marked viscosity, with intermittent release, in drops or ejaculation of small quantities, with very

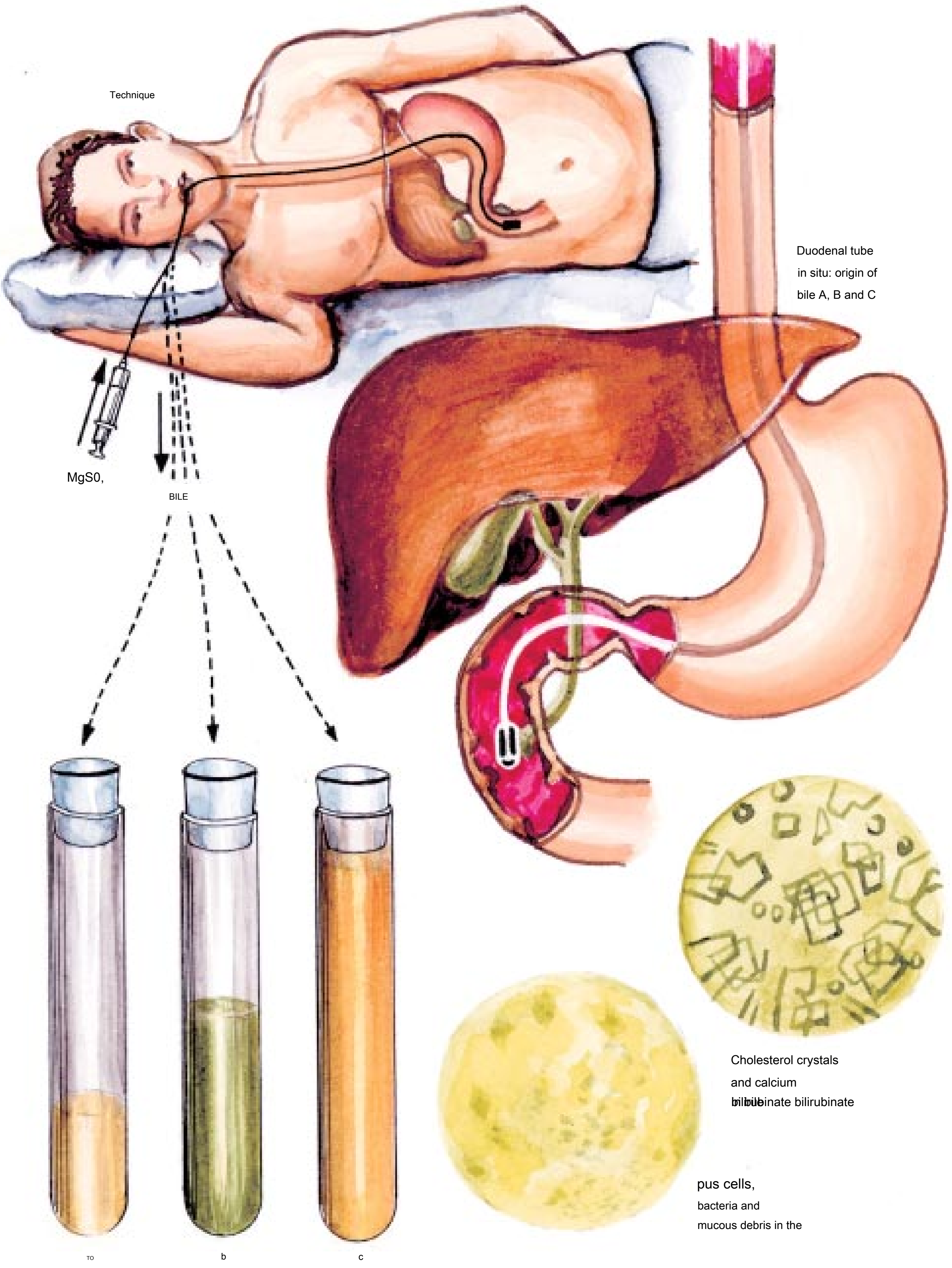

Technique
Duodenal tube
in situ: origin of
bile A, B and C
MgS0,
BILE
Cholesterol crystals
and calcium
bilirubinate bilirubinate
pus cells,
bacteria and
mucous debris in the
TO
b
c

Fig. 62.35 Normal data and techniques of biliary drainage.

few bile-stained flocs. It comes from the gallbladder. On microscopic examination, leukocytes, mucus, bile-stained epithelium, and some bacteria appear.

Bile C. It is obtained in an amount of 80-100 mL; It is fluid, straw yellow and gold in color, with a continuous flow, with some flocs. It is of hepatocanalicular origin.

Pathological data

1. Difficulties in duodenal catheterization.

These arise because the tube does not pass into the duodenum. They are presented in:

a) Pylorus spasm.
b) Gastric ptosis.
c) Cascade stomach.

2. Alterations of bile samples:

a) Bile alterations A.

Permanent absence of bile A. One should think about permanent biliary obstruction, generally produced by cancer of the head of the pancreas with obstruction of the common bile duct, cancer of the bile ducts, ampullomas, foreign bodies, stones, parasites, etc., which create obstruction.

Transient absence of bile A. Corresponds to obstructions of an inflammatory nature, which are reversible, as occurs in gallstones, choledocytis, spasms of the common bile duct and sphincter of Oddi, chronic pancreatitis, etc. In acute hepatitis, an absence of bile is observed during the first days, which then reappears in the following days.

Bile A, pseudo B. In these cases bile A., constituted fundamentally by hepatic bile Co, is highly concentrated similar to bile B, without actually coming from the gallbladder, it comes from the bile channels where a phenomenon of stagnation and hyperconcentration has occurred. This type of pseudo B bile is observed in cases of canalicular dilation secondary to gallbladder absence or obstruction. Pseudo B bile is also observed in cases of pleiochromia (hemolytic jaundice).

b) Alterations of vesicular bile.

Absence of bile B (null vesicular response). It expresses manifest alteration of the gallbladder with loss of its function, either due to occlusion of its neck, or of the cystic, or because the individual has undergone a cholecystectomy, or due to congenital absence of the gallbladder organ. The most frequent or important possibility of absence of bile is permanent gallbladder blockage, which It occurs due to multiple causes, the most frequent being: calculous cholecystitis, gallbladder cancer, empyema, hydrocholecystitis, atrophic cholecystitis, strawberry gallbladder or cholesterolosis and intense pericholecystitis.

Alterations in the quantity and quality of bile B. When vesicular bile B is altered, whether in quantity or quality, what is known as gallbladder functional capacity is lost and thus when bile B is not obtained we speak of null functional capacity and if the quantity is reduced, we speak of mediocre or diminished functional capacity, which is the case at hand. An increase in the amount of bile B is observed, which can sometimes reach 100 mL; 150, 200 mL and more in cases of atonic vesicular dyskinesia or cholecystoatonia. A decrease in the amount of bile B (less than 50 mL) can be observed in calculous cholecystopathies or not, as long as the gallbladder has lost its functional capacity.

The color of bile B is also pathologically altered; and offers a dark color in hyperconcentration due to stasis, a light color in hypoconcentration (poorly concentrating gallbladder) and green color in cases of biliary sepsis (minty bile).

c) Alterations of hepatic bile Co. It may be increased (hypercholeresis) or decreased (hypocholeresis); If the color is brown, amber or tobacco, this bile can be confused with bile B; It is the so-called pseudo B bile.

d) Microscopic alterations.

Microscopic examination of bile flocs allows the following elements to be studied:

Duodenal epithelium. It is made up of large cells, twice as large as a leukocyte; oval or cuboidal in shape, pearl gray in color and may be stained by bile; its alterations are observed in duodenal inflammatory processes.

vesicular epithelium. Its cells are long columnar and appear in the shape of a fan or rosette, forming groups; heavily stained by bile. They are an index of gallbladder exfoliation and are present in cases of cholecystitis, sometimes accompanied by leukocytes, pyocytes and bacterial colonies.

Bacterial colonies. In the form of cocci, chains, etc., they indicate infection. Fungal mycelia can also be observed.

Mucus. It is stained by bile, when it appears in the form of bands; It is more specific to the bile ducts, especially the cystic duct.

Oily substance. So called because of its similarity to oil, it indicates cystic catarrh.

Cholesterol crystals. They appear transparent, rectangular in shape, of various sizes and are an indication of cholesterin lithiasis.

Calcium bilirubinate crystals. They are golden yellow or reddish in color, of various sizes. They generally appear concomitantly with cholesterol crystals, which constitutes the so-called lithogenic material, quite suspicious of gallstones of gallbladder location if it predominates in the bile B sample.

High columnar epithelium (of common bile duct). Similar to that of the vesicle, but less stained.

Canalicular epithelium Polygonal and highly stained with bile; It is rare.

Hematies and leukocytes. With well-known characteristics, they indicate hemorrhage or infection in the location identified by the epithelium found.

Parasites. Giardia lamblia, histolytic amoeba, hepaic fasciola, Clonorchis sinensis, Strongyloides estercolaris, among others, whose characters have been studied in Parasitology.

Timed bile drainage

This test records the quantity of the different fractions of bile by measuring their appearance time, duration and amount of flow. The presence of pain (if it exists) is also noted in relation to the period in which it appears.

Elimination of bromosulfthalein (BSF) in bile

It corresponds to the time of appearance of BSF in the bile according to Carolí's technique. Measuring the time of appearance The presence of BSF in the bile allows us to differentiate, in case of retention of the dye in the blood, two types; different pathophysiological conditions in the presence or absence of jaundice.

Retention due to parenchymal injury will give a normal or decreased appearance time of BSF in the bile.

Retention due to obstruction of the main bile duct is recognized by a prolonged onset time.

The normal appearance time ranges between 15 and 20 min. It is decreased in hepatitis and cirrhosis, and increases, exceeding 25 min, in intrahepatic cholestasis but more so in extrahepatic cholestasis.

EXPLORATION OF PANCREATIC FUNCTION

Duodenal intubation and exogenous stimulation with secretin and pancreozymine constitute the most sensitive and specific test to evaluate primary alterations of exocrine pancreatic function.

After placing the probe in the duodenum, the stimulation with these hormones is carried out, and the total volume of pancreatic juice is studied, as well as its concentration in bicarbonate and enzymes (amylase and lipase). Secretin stimulates bicarbonate-rich secretion, while pancreozymin stimulates enzymatic secretion.

The results obtained allow a diagnosis of exocrine pancreatic insufficiency to be made. In chronic pancreatitis, enzyme secretion is more intense and altered earlier.

Normal data

Normal data for pancreatic catheterization are:

Bicarbonate	60 -140 mEq/L
Lipase	30 U Biondi (bile):
	0.2 U (in serum) 80 -
Serum Amylase	18OU 100-
Volume	150 mL

The interpretation of the results of pancreatic catheterization should be carried out with the help of Table 62.

Table 62.1

Vol n1	Bicarbonate	Enzymes	Diagnosis
Diminished	Normal	Normal	Pancreatic duct obstruction
Normal	Diminished	Decreased	Chronic processes of the pancreas
Increased	Diminished	Normal	Hemochromatosis
Normal	Normal	Normal	No exocrine pancreatic involvement

OTHER STUDIES USING CATTERS OR PROBES

Esophageal manometry

The objective of this test is to analyze esophageal motility. It is the test of choice along with barium studies for the diagnosis of motor disorders of the esophagus. It also enables the physiological evaluation of the lower esophageal sphincter by studying the pressure variations that occur at its level, which is very useful in the evaluation of gastroesophageal reflux disease, where alterations in the resting pressure and the adaptive pressure changes of this sphincter play an important role in the pathophysiology of this disease.

The test consists of detecting intraesophageal pressures using intracavitary microtransducers placed at the distal end of a probe.

There are also methods that use extracorporeal pressure transducers, which receive pressure changes from the esophageal lumen, through catheters with a constant perfusion of distilled water or isotonic saline solution, thus creating a continuously perfused chamber that is only open at its distal end, through which the liquid flows freely.

Intraesophageal pressure changes have an impact on the catheter orifice, compromising the output of the perfused liquid, which ends up having an impact on the transducer, which transforms these pressure changes into an electrical signal and is transmitted to a recording system.

In the case of intracorporeal (intracavitary) microtransducers, the pressure chambers have been miniaturized, and are introduced together with the catheter into the esophagus, with the advantage of not needing a hydraulic system to transmit pressure variations, since these are transformed in situ into an electrical signal.

Pathological data

Normal data related to esophageal anatomy and physiology were discussed in Chapter 56, studying dysphagia.

The esophageal dyssynergia syndrome is characterized by the existence of a high base pressure, absence of pressure peaks corresponding to peristaltic waves and disappearance of the decrease in said pressure through the opening of the cardia.

In esophageal spasms, the existence of abnormal areas of high pressure is recorded.

In scleroderma, the existence of a line without undulations reveals the lack of contractile activity in the lower two-thirds of the esophagus.

When the lower sphincter is injured, the wave indicating the positive pressure generated by it disappears.

esophageal ph-metry

Esophageal pH-metry is the technique that allows the study of gastroesophageal reflux by measuring the intraluminal pH of the esophagus.

Under normal conditions the esophagus maintains a more or less neutral pH. By having pH electrodes, which due to their small size can be placed at the distal end of a fine catheter and introduced into the esophagus, we then have a test that can detect the existence of gastroesophageal reflux through the pH variations that occur in the esophageal lumen.

The development of small portable instruments, which can be carried with the patient, has allowed the monitoring of pH for 24 hours, under a lifestyle that attempts to approximate the usual one. This test is the most effective in clinical practice for the diagnosis of gastroesophageal reflux disease. However, these studies should be reserved for particular situations in which their use may require a diagnosis that has not been evident by other methods, since the majority of patients with gastroesophageal reflux disease do not require the study of intraesophageal pH because they present classic symptoms, which are sufficient to establish the diagnosis, or in whom the presence of esophagitis has been demonstrated by endoscopic methods.

Blind gastric biopsy

In this test, the Crosby or Watson probe is used, which has a capsule at its distal end, which consists of a rotating blade activated by a spring. This blade cuts the mucosa fraction sucked by a syringe that fits the proximal end of the probe.

The biopsy obtained in this way provides good information in diffuse diseases of the stomach.

Peroral biopsy of the small intestine

It is the only method capable of ensuring the integrity of the mucosa for study. Its performance is essential in any patient with suspected diffuse enteropathy, and is part of the battery of tests to study a patient with malabsorption syndrome.

The mucosa sample can be obtained using any of the capsule probes, which work with an aspiration-section mechanism. The most commonly used probes are Crosby and Watson.

Colonic manometry

From a technical point of view, the procedure is based on the same principles that we refer to in esophageal manometry, open-end catheters with continuous perfusion that transmit pressure variations to an external transducer.

The currently known colonic motor alterations consist of quantitative variations in the components that make up the normal motor pattern, and specific colonic motor patterns have not yet been described, in contrast to what happens in the upper digestive tract: therefore, the diagnosis of colonic motor disorders still rests on the basis of their symptoms and not on the results of motor studies.

Finally, we must mention other examinations such as: electrophysiological studies (the description of which is beyond our objectives) and gastric cytological study.

The gastric cytological study consists of the microscopic analysis of the exfoliated cells of the gastric mucosa, which once obtained are spread on a slide, fixed in alcohol-ether and then colored by the Papanicolau technique (1941). The cells appear isolated or in groups, which allows the early diagnosis and discovery of the development of a malignant tumor due to the characteristics of the cells examined, such as: alterations in the structure of the nucleus, which make it appear in the form of large clumps, with free spaces between them.

In order to obtain cells for cytological study, different means have been used such as: gastric abrasive balloons, which are covered by a mesh (like a woman's hairnet) in which the cells are imprisoned; Ayre's otatory brush (1953), which consists of brushes designed by this author, which are rotated from the outside, in order to sweep the mucosa, and to trap the cells between the bristles of the brush.

Gastric lavages with a buffer solution of papayin or chymotrypsin are also used, which cause desquamation by dissolving the mucus; The extracted liquid is centrifuged and then smears are made with the sediment. Sometimes, if no other means are available, gastric lavage can be done simply with saline solution or Ringer's solution.

When solutions are used to collect desquamation cells, it is advisable, once the liquid is in the stomach, to have the patient adopt several positions:
face up, on the right side, face down, on the left side and finally face up so that the liquid bathes all the walls of the stomach and thus the best possible material is collected.

MAIN SYNDROMES OF THE DIGESTIVE SYSTEM

63

Introduction

In this chapter we will deal with the main syndromes of the digestive system; To begin, we will deal with gastrointestinal hormones, since they can give rise to various syndromes in the functional order and also cause tumors that are known as apudomnas.

Ulcer syndrome and acute abdominal syndromes will not be treated in this book, since they are widely studied in Internal Medicine and Surgery.

Below we will list the topics that are the subject of our study:

- Gastrointestinal hormones.
- Jaundice syndrome.

Ascitic syndrome

- Chronic diarrheal syndromes.

Digestive bleeding.

GASTROINTESTINAL HORMONES

The role that the digestive system plays as an endocrine organ has been known for years, but it was at the end of 1960 that a series of new discoveries of substances with characteristics of true hormones secreted by the gastrointestinal tract took place. Of these hormones, the best known are: gastrin, secretin, cholecystokinin or pancreocymin, insulin and glucagon.

Another group of substances with some characteristics of hormones, but that do not yet meet all the requirements to be considered as such, are: vasoactive intestinal polypeptide, gastric inhibitory polypeptide, motilin, enteroglucagon, pancreatic polypeptide, and others.

In 1968, the term APUD was introduced to refer to cells whose main function is the production of hormones and that have common cytochemical and ultrastructural characteristics. The term APUD derives from the initial letters in English related to the characteristics amine transformers that the cells of this group have: Amine Precursor Uptake Decarboxylation (precursors of the uptake and decarboxylation of amines).

In later years, the endocrine cells of the digestive system have been classified and named with the letters G, D, S, E, etc.; The tumor processes that have their origin in these cells, as we already said, are called apudomas.

Below we will briefly explain the best-known hormones of the digestive system.

GASTRINE

In 1905 gastrin was discovered by Edkins, but it was not until 1964 that it was purified for the first time. Several types of this hormone have been described according to the number of amino acids that make it up. The most widely used form of synthetic gastrin is pentagastrin, which is used as a clinical test of gastric secretion.

Gastrin is secreted mainly by cells called G located at the level of the gastric antrum, although it can also be produced at the level of the duodenum.

It is released by stimulation of the vagus nerve and also by distension of the anthropyloric region of the stomach, and by contact of food with the mucosa.

The main action of gastrin is the stimulation of acid secretion from the stomach.

It also stimulates gastric and intestinal motility and contraction of the lower esophageal sphincter.

The existence of hypergastrinemia has been demonstrated in some patients with duodenal ulcer and in Zollinger-EIlison

SECRETIN

First hormone discovered in a duodenal extract.

This event, attributed to Baylis and Starling, occurred in 1902.

But it was not until 1961 that this hormone was isolated and its structure was determined. In 1966 it was synthesized for the first time.

Secretin is secreted mainly in the duodenum by the so-called S cells, which have also been found in a smaller proportion in the jejunum.

Its release occurs mainly due to the arrival of acid chyme to the duodenum or jejunum. To a lesser extent, proteins, fatty acids and other ingredients of gastric chyme also stimulate its release.

Its main action is the stimulation of the secretion of water and bicarbonate by the pancreas ("hydrolatic" secretion), which is why it is used in tests of pancreatic function. Among other actions it has are: inhibition of acid secretion of the stomach and its motility; it strengthens the action of the cholecystokinin on vesicle contraction and stimulates insulin secretion.

CHOLECYSTOKININE

In 1928 Ivy and Goldberg observed that when fat entered the small intestine it stimulated the release of a substance that caused the gallbladder to contract: they called this substance cholecystokinin.

In 1943, Hasper and Raper discovered a hormonal-like substance in the mucosa of the duodenum that stimulated the enzymatic secretion of the pancreas and they named it pancreocymin.

As biological studies progressed and both hormones were purified, it became clear that they were actually the same substance; Currently the accepted name for this hormone is cholecystokinin.

This is secreted by the so-called I cells that are found mainly in the duodenum and jejunum.

It is released by the presence of amino acids, fatty acids and hydrochloric acid.

Its fundamental actions are: stimulation of the enzymatic secretion of the pancreas ("ecbolic" secretion) and the contraction of the gallbladder. It is used in clinical practice for functional studies of the pancreas and biliary tract. It also has effects on the motility of the stomach and intestine, and in large quantities stimulates acid secretion from the stomach.

INSULIN

It is secreted by B cells located in the center of the pancreatic islets. Its secretion is induced by different factors, first of all by glucose. Among the hormonal factors that stimulate insulin secretion are: ACTH, the growth hormone, glucocorticoids, estrogens and progesterone.

Insulin is the great hormone of anabolism, it increases the penetration and use of glucose by peripheral tissues; It is hypoglycemic and lipogenic; It promotes cellular penetration of amino acids and potassium, protein synthesis and water retention.

GLUCAGON

It was discovered in 1923 and first synthesized in 1968. It is secreted by the islet cell of Langerhans.

The factors involved in its release are essentially metabolic: glucose, amino acids and plasma levels of free fatty acids.

Its fundamental action is hyperglycemic, stimulates glycogenolysis and promotes glucose uptake in muscle and adipose tissue.

It is an emergency hormone, since it increases the circulating rate of elemental nutrients and stimulates the release of insulin and adrenaline.

The effects it has on the gastrointestinal tract are used for diagnostic and therapeutic purposes. These effects are: it inhibits the production of stomach acid, dilates the duodenum, suspends the excretion of enzymes and pancreatic juice, and relaxes the muscles of the small and large intestine.

GASTRIC INHIBITOR POLYPEPTIDE

It was isolated and purified in 1969, from extracts of duodenal and jejunal mucosa of pork; Its release is mainly stimulated by meals.

It has a powerful inhibitory effect on gastric secretion and also stimulates the release of insulin.

MOTILIN

In 1966 it was discovered that when the dog's duodenum was alkalized, powerful contractions were produced in the isolated intestine and in the fundic area of the stomach, which suggested the participation of a hormone in this phenomenon. It was isolated in 1971 and was called notilin.

It is secreted at the level of the small intestine, especially in the jejunal and duodenal mucosa and the proximal portion of the ileum. It is released by intense acidity and after a meal.

In addition to being a powerful stimulant of intestinal motor activity, motilin increases pepsin production and inhibits gastric emptying. It can also cause pressure changes in the lower esophageal sphincter and could be important in the pathophysiology of gastroesophageal reflux.

PANCREATIC POLYPEPTIDE

It is secreted by DI cells located on the periphery of pancreatic islets. Its release is stimulated after a meal.

Its main effects are opposite to those of cholecystokinin, including inhibition of gallbladder contraction, inhibition of enzyme secretion from the pancreas, and increased tone of the main bile duct. It inhibits gastric and intestinal motility and stimulates acid secretion from the stomach and bicarbonate secretion from the pancreas.

VASOACTIVE INTESTINAL POLYPEPTIDE

It was isolated in 1972 from extracts of animal small intestine as a substance that produces peripheral vasodilation.

It is secreted throughout the digestive tract, with predominance in the small intestine, by cells called H.

It has a wide variety of effects including: vasodilation, inhibition of gastric acid and pepsin secretion, stimulation of hydrocarbon secretion of the pancreas and bile secretion; Like glucagon, it produces hyperglycemia.

This peptide is considered responsible for the symptoms present in Verner-Morrison syndrome: watery diarrhea and insular cell adenoma of the pancreas.

ENTEROGLUCAGON

In 1961, cells with glucagon activity were discovered in the intestine and the substance produced there was called enteroglucagon, which differs from pancreatic glucagon due to its antigenicity and biological and physicochemical properties, although its exact structure is not known.

It has a glycogenolytic activity lower than that of pancreatic glucagon and insulinogenic and lipolytic actions have also been reported.

Another group of substances still under study, since they have been isolated in animals but not in man, and that are considered as possible hormones includes: enterogastronas (inhibit acid secretion of the stomach), bombesin (stimulates gastric and pancreatic secretion and vesicular contraction) and villikinin (stimulates the motility of intestinal villi).

The better study of these substances and hormones, and the exact knowledge of their structure and properties, will allow in the future to better understand a series of gastrointestinal disorders whose exact etiology is still unknown and could probably explain many of the disorders of the digestive tract called functional today.

ICTERIC SYNDROME

DEFINITION AND CONCEPT

Jaundice or jaundice (from the Greek ictus: yellowness) means the yellow coloring of the skin, mucous membranes and some organic fluids, which is due to their impregnation by abnormally increased bilirubin in the plasma (hyperbilirubinemia). Jaundice is rather a syndrome in which the yellow discoloration of the skin and mucous membranes stands out as the fundamental symptom.

Three questions arise when faced with yellowish discoloration of the skin and mucous membranes:

1. Is it jaundice? (Positive and differential diagnoses.)

2. If it is jaundice, what is its production mechanism? (Pathogenic diagnosis.)
3. What is its etiology? (Etiological diagnosis.)

SYNDROMOGRAPHY 0 POSITIVE DIAGNOSIS

Clinical picture

The most striking symptom is the yellow color of the skin and mucous membranes: there is a true impregnation in these tissues, so that if the icteric skin is compressed with a slide, this coloration persists and becomes more evident. This coloration also exists in the deep visceral organs where there are very related organs, such as the heart valves, and others that are not very related, such as the cornea, muscles and the nervous system; It is curious that the cerebrospinal fluid is practically not stained, even in cases of very intense uterus.

The intensity varies from barely visible jaundice, called subicters, to frank jaundice.

In the study of the jaundice symptom (see semiotechnics and semiography in volume 1, Chapter 25) we have detailed the fundamental characteristics. We will point out here only the most important elements of the jaundice syndrome.

During the inspection, the place or region where the jaundice is most visible should be looked for: sclera, underside of the tongue, palatine vault, soft palate, skin of the forehead, chest, abdomen and inner thighs.

According to its tone or tint, it is classified into:

flavinic, rubinic, verdinic and melanic.

Among the associated symptoms that give more value to this yellow coloration to interpret it as true jaundice, we must mention itching, which is often manifested by scratching marks, but other times it is more intense, as observed in obstructive jaundice.

Another objective symptom is xanthelasma, which is attributed to a cholesterol deposit.

Among the digestive symptoms we have anorexia, hypocolia, or acholia when the jaundice is obstructive, or fecal pleiochromia when it is a hemolytic jaundice.

The urine is light or dark brown in color, called choluria, and when shaken it forms a yellow foam due to the presence of bile pigments. In hemolytic jaundice there is only urobilinuria, which forms a white foam.

Cardiovasculohematic symptoms include sinus bradycardia, arterial hypotension, and hemorrhages that occur in severe jaundice due to prothrombin deficiency, or in obstructive jaundice totals due to vitamin K absorption deficiency, which also leads to a prothrombin deficiency.

Among the nervous symptoms we have insomnia, asthenia and visual disorders: hemeralophia and xanthopsia.

Complementary exams

We corroborate the diagnosis of jaundice by measuring the bile pigments in the blood; Depending on the amount of circulating bilirubin, jaundice can be:

Latent jaundice. Circulating bilirubin increases, but without clinical manifestation (they are simple hyperbilirubinemias).

Subictero. Bilirubin of 1-1.5 mg%.

Frank jaundice. There are more than 2 mg %.

Both in jaundice due to liver disorders and in obstructive jaundice (due to excretion disorders) the percentage of direct or conjugated bilirubin (monoglucuronide or diglucuronide) is elevated, while in hemolytic jaundice the indirect or unconjugated bilirubin is elevated.

We must also carry out a study of the pigments in feces and urine, as well as the so-called liver function tests, which we will point out later.

DIFFERENTIAL DIAGNOSIS

We will have to differentiate jaundice from yellowish discolorations that are not due to an increase in bilirubin, and which constitute the so-called pseudojaundices. Among them we have those that follow the ingestion of picric acid, foods rich in carotene, or atebrin.

In these cases:

a) There is no pigmentation of the mucous membranes or sclera.

b) There are no pigments or bile salts in the urine.

c) The amount of bilirubin in the blood is normal.

It is easier to make the differential diagnosis with:

a) The straw yellow color of subjects affected by cancer.

b) The greenish-yellow tint of chlorotic patients.

c) The waxy yellow color of pernicious anemia.

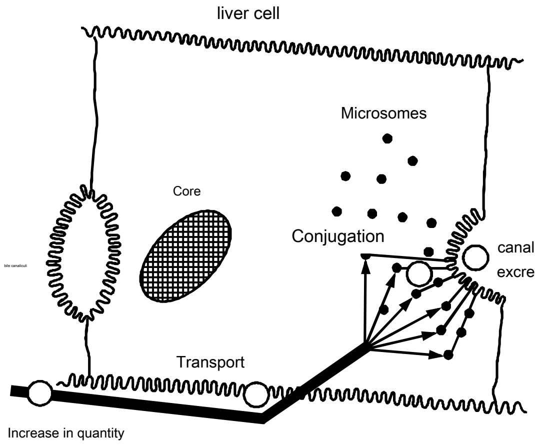


Fig. 63.1 Pathophysiology of the jaundice syndrome.

SYNDROMOGENESIS 0 PATHOPHYSIOLOGY (PATHOGENIC DIAGNOSIS) (FIG. 63.1)

Jaundice, according to the mechanism that produces it, can be:

- Jaundice due to increased production of free or unconjugated bilirubin that reaches the liver cell.
- Jaundice due to disorders in the uptake of free bilirubin by the hepatocyte.
- Jaundice due to disorders in the conjugation of bilirubin within the hepatocyte.
- Jaundice due to disorders in the excretion of conjugated bilirubin from the hepatocyte to the duodenum.

jaundice due to increased production of free or unconjugated bilirubin that reaches the liver cell

Within this group we can distinguish:

1. Hemolytic jaundice. They are due to a marked increase in the destruction of red blood cells, which results in excessive formation of indirect bilirubin at the expense of the released hemoglobin (Fig. 63.2). This fact does not necessarily mean anemia, which only occurs when erythrocyte destruction is such that the erythropoietic reserve of the bone marrow is incapable of counteracting it.

2. Short circuit hyperbilirubinemia. In these cases, jaundice is not always detected clinically. They originate from the production and increase of bilirubin from hemoprotein in the bone marrow.
3. Dyserythropoietic jaundice. They are due to the premature destruction of erythrocyte precursors during their maturation phase.

Clinical picture

The clinical picture of jaundice due to an increase in the production of free bilirubin will depend on the cause of the jaundice. In those caused by short circuit and in dyserythropoietic ones, as we already said, jaundice is barely detectable clinically. There may be paleness, asthenia, palpitations and in some types of hemolytic jaundice with anemia, rheumatic pain and abdominal painful crises may occur. The tint in hemolytic jaundice is light yellow (flavinic).

Complementary exams

We find an increase in unconjugated bilirubin, but there is no choluria because this bilirubin is not filtered through the renal glomerulus. However, there is an increase in urobilin, as well as urobilinogen or fecal stercobilinogen, which is responsible for pleochromia.

The hematological picture is of great value, since significant anemia can be detected that will help specify its etiology.

jaundice due to disorders in the uptake of free bilirubin by the hepatocyte

Here the defect lies in the transport of free bilirubin from the plasma to its conjugation site in the liver cells (smooth endoplasmic reticulum). The best example of this group is Gilbert's disease, which is a familial congenital nonhemolytic jaundice (Fig. 63.3).

This disorder can also occur after viral hepatitis or in the newborn.

In its intimate mechanism, a deficiency of cytoplasmic proteins (I Z) of the hepatocyte has been considered, which would play an important role in the uptake and transport of bilirubin to the microsomes where conjugation takes place.

Clinical picture and complementary tests

They are similar to those described in the previous group. If there is a simultaneous increase in conjugated bilirubin, it may reflect a certain degree of liver dysfunction.

jaundice due to disorders in the conjugation of bilirubin within the hepatocyte

The conjugation of bilirubin, as we said previously, takes place in the microsomes of the liver cell, where the enzyme glucuronyltransferase plays a primary role. Following the pathogenic mechanisms that can affect conjugation, we can subclassify these jaundices into three groups:

1. Due to enzymatic immaturity.

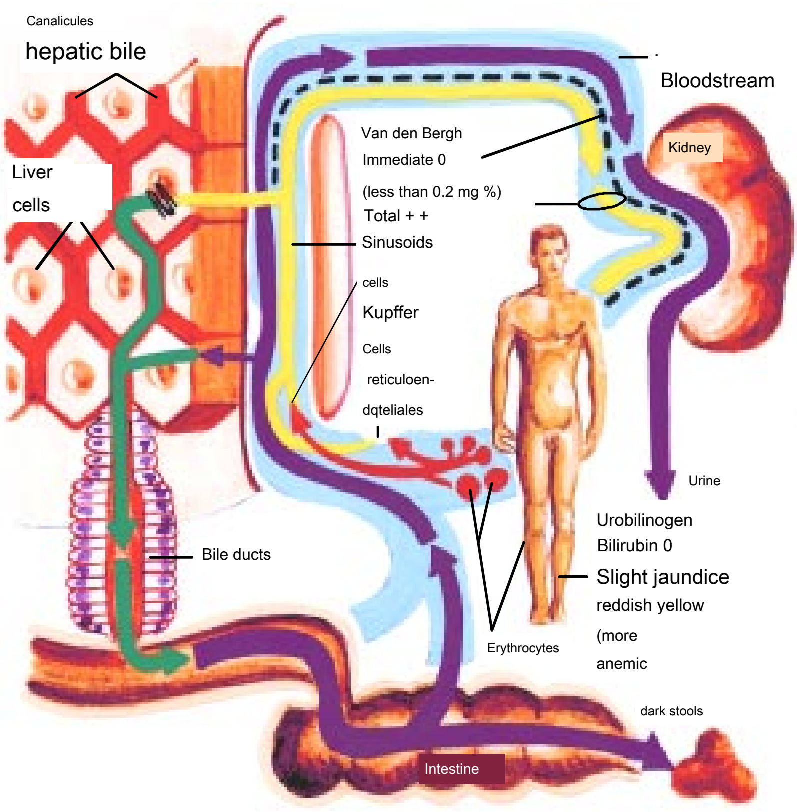

Canalicules
hepatic bile
Liver
cells
Bloodstream
Van den Bergh
Immediate 0
(less than 0.2 mg %)
Total + +
Sinusoids
cells
Kupffer
Cells
reticuloen-
dqteliales
Kidney
Urine
Urobilinogen
Bilirubin 0
Slight jaundice
reddish yellow
(more
anemic
Bile ducts
Erythrocytes
dark stools
Intestine

Fig. 63.2 jaundice due to increased bilirubin production (hemolytic).

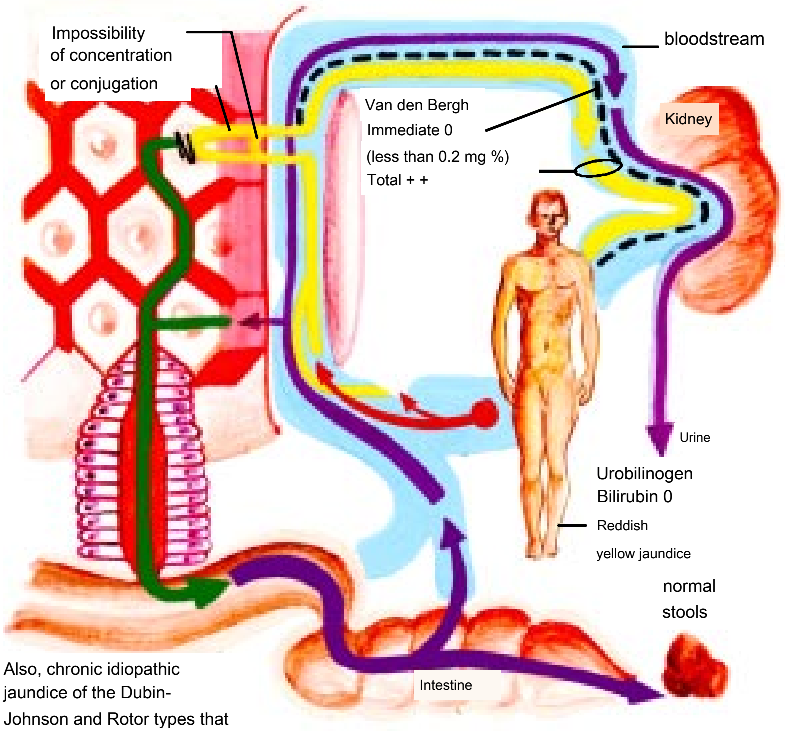
KEY

🟥 Biliverdinglobin

🟨 Indirect bilirubin (unconjugated)

🟩 Immediate reaction bilirubin (conjugated)

🟪 Urobilinogen


Impossibility
of concentration
or conjugation
bloodstream
Van den Bergh
Immediate 0
(less than 0.2 mg %)
Total + +
Kidney
Urine
Urobilinogen
Bilirubin 0
Reddish
yellow jaundice
normal
stools
Intestine

Also, chronic idiopathic jaundice of the Dubin-Johnson and Rotor types that present immediate reaction

Fig. 63.3 Jaundice due to uptake disorders.

2. Due to absence or congenital deficiency of glucuronyl transferase.

3. By inhibition of glucuroconjugation.

The first group includes the so-called physiological jaundice of the newborn and premature infants, which is due to a temporary insufficiency of the enzyme glucuronyltransferase. Certain factors can favor these jaundice such as: hypoxia, hematomas, hypoalbuminemia, as well as the administration of certain medications (sulfamidates, salicylates and vitamin K).

In the second group, due to the absence or congenital deficiency of glucuronyltransferase, is Crigler-Najjar disease, a familial jaundice that generally appears in the first days of life.

In the third group, due to inhibition of glucuroconjugation, substances from the pregnandiol group have been described in the serum and mother's milk, which are capable of inhibiting the conjugation of bilirubin. A similar effect can be produced by certain medications such as novobiocin.

Clinical picture

It is transient and not very fluid when the disorder is due to transient deficit or inhibition, being much more serious in Crigler-Najjar, which can reach high levels of indirect bilirubin and develop a kernicterus (impregnation of the gray nuclei at the base of the brain).
Children affected by this disease usually die in the first year of life, although survival into adulthood has been described.

Complementary exams

As in the previous groups, we will find an increase in unconjugated bilirubin.

jaundice due to disorders in the excretion of conjugated bilirubin, from the hepatocyte to the duodenum

Jaundice due to disorders in bilirubin excretion can be divided into two groups: intrahepatic cholestasis and extrahepatic cholestasis, according to the location of the obstruction (Fig. 63.4) .

Intrahepatic cholestasis (Fig. 63.5)

Intrahepatic cholestasis is understood to be biliary stasis in which there are no demonstrable obstacles in the extrahepatic bile ducts. Some authors classify intrahepatic cholestasis as mechanical and functional, grouping among the former those due to an obstacle in the intrahepatic bile ducts, such as: cholangiocarcinomas, atresia of the intrahepatic bile ducts, metastases, etc.

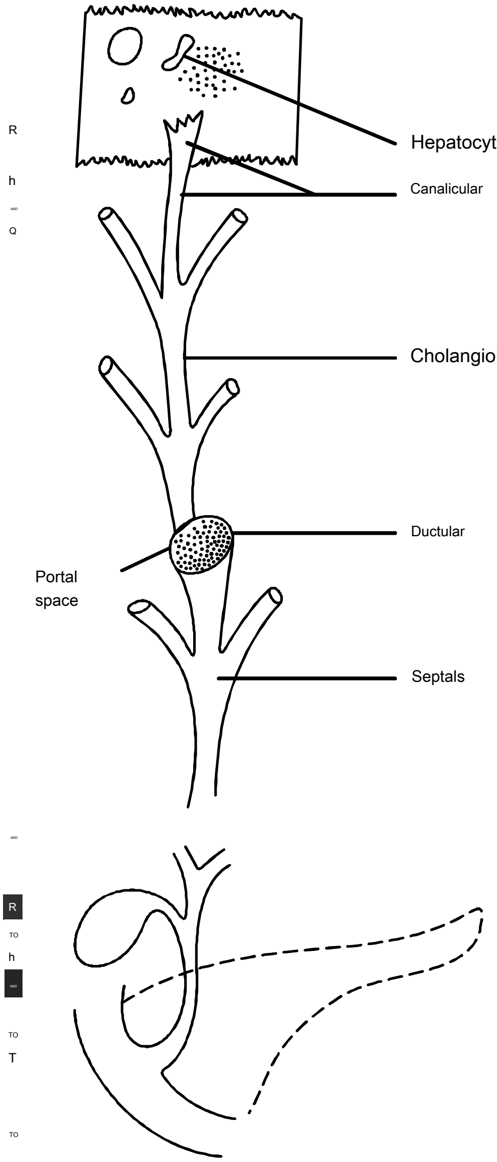


Fig. 63.4 Types of cholestasis: intrahepatic and extrahepatic.

Intrahepaic cholestasis without an obstructive cause is precisely the most frequent and its exact mechanism of production is still unknown. They can be subdivided into: hepatocytic, canalicular, cholangiolar, ductular and septal, according to the site of the intrahepatic bile ducts where the cholestasis lies (see Fig. 63.4).

Within the group of canalicular jaundices, hepatitis and cirrhosis deserve a separate paragraph (Fig. 63.6), which are the most frequent and which some authors include within a group called hepatocellular jaundices, since the mechanism of their production is more complex.

Necrosis of liver cells causes bile excreted through the bile canaliculi to pass through the sinusoid into the blood and mix with it. Even without complete cell necrosis, ruptures of some cell membranes may occur and as a consequence

Due to them and bile stasis, fissures are produced through the cellular cytoplasm, establishing communications between the biliary system and the blood.

In both cirrhosis and hepatitis, due to profound alterations in the lobular structure and disorders of liver function, the processes of uptake, conjugation and excretion of bilirubin can also be altered.

Clinical picture

The clinical characteristics can be very varied, from almost asymptomatic, as is the case with some drug-induced jaundice, to florid pictures of liver cirrhosis with all its symptomatic procession and multiple physical signs, which occur in addition to jaundice, such as hepatomegaly, splenomegaly, ascites, telangiectasias, edema in the lower limbs, etc.

Complementary exams

In laboratory tests, an increase in both conjugated and unconjugated bilirubin can be found, which depends on the degree of cholestasis and liver failure.

We will also find an increase in glutamicoxalacetic transaminase and especially pyruvic transaminase, prolongation of prothrombin time, positivity of flocculation tests (thymol), etc.

To specify the diagnosis, it is very useful to perform a laparoscopy and a liver biopsy, which will give the typical anatomical picture of hepatitis and cirrhosis, and will specify the degree and level of cholestasis.

Extrahepatic cholestasis (Fig. 63.7)

In extrahepatic cholestasis, the mechanism of jaundice production is always demonstrable, since there is an obstacle to the flow of bile from the liver to the duodenum, due to multiple causes (stones, neoplasms, extrinsic compressions, etc.).

In cholestasis of this type, also called obstructive, the mechanism of production is very easy to understand, since when there is an obstacle in any part of the extrahepaic biliary tree, this implies a marked increase in pressure in the bile ducts that when it reaches a certain level breaks the intrahepatic bile canaliculi, allowing communication with the sinusoids and the passage of bile into the blood.

Maintained stasis also injures and necroses many liver cells, contributing to creating more communications between the biliary and vascular trees.

Clinical picture

Extrahepatic cholestasis generally presents in middle or advanced age, suddenly or insidiously, with chills and fever, or anorexia or cachexia and intense hepatic colic or abdominal pain. The general condition in benign cases is good, on the other hand

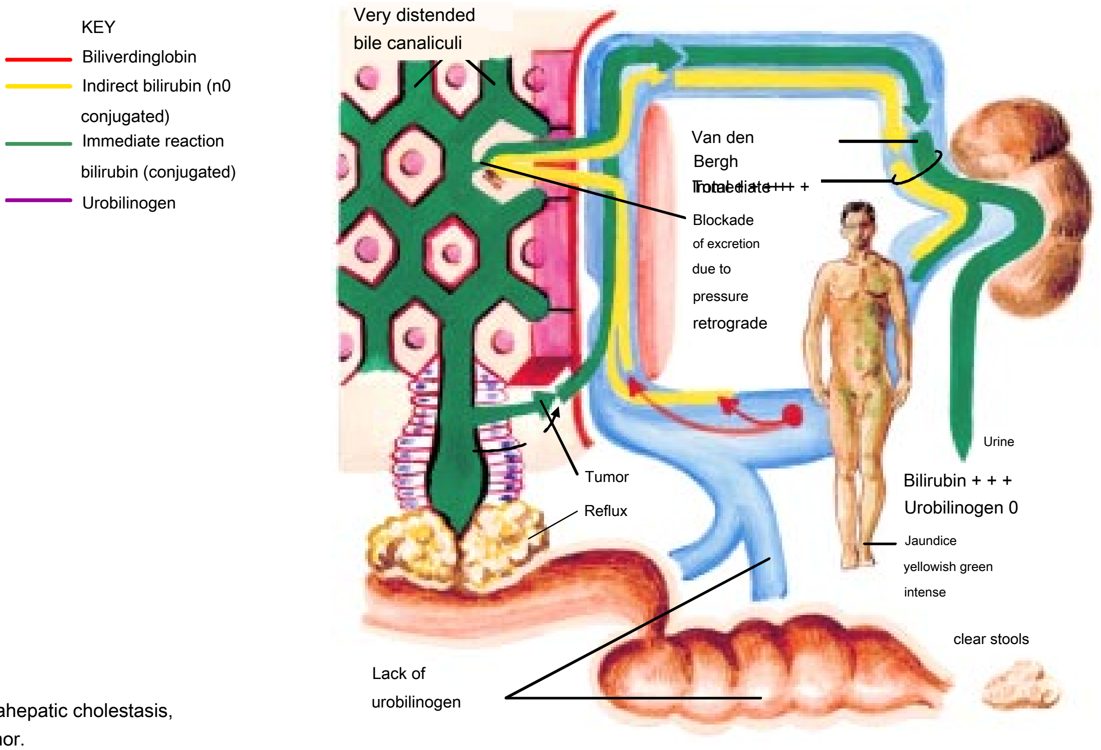

KEY
🟥 Biliverdinglobin
🟨 Indirect bilirubin (n0 conjugated)
🟩 Immediate reaction bilirubin (conjugated)
🟪 Urobilinogen
Very distended bile canaliculi
Van den Bergh
Total + + +
Immediate +
Blockade of excretion due to pressure retrograde
Tumor
Reflux
Urine
Bilirubin + + +
Urobilinogen 0
Jaundice yellowish green intense
clear stools
Lack of urobilinogen

Fig. 63.7 Complete extrahepatic cholestasis, for example, from a tumor.

bio in malignant cases, the patient appears serious;

After a certain time, the jaundice takes on a greenish or melanic color; the liver is generally enlarged by cholestasis; the normal gallbladder, small or large;

the spleen normal; The itching may be intense with large scratching lesions.

Complementary exams

Bilirubin reaches high levels, especially at the expense of direct or conjugated bilirubin.

Liver flocculation tests are frequently negative, except in prolonged cases.

Cholesterol and alkaline phosphatase are elevated, as are phospholipids.

Prothrombin time is prolonged due to lack of vitamin K absorption.

There is acholia in complete obstructions and the choluria is intense; However, there is no urobilinuria, since stercobilinogen is not formed as bile does not flow to the intestine.

Radiological and laparoscopic studies are of value, and in some cases laparoscopic cholangiography, to specify the etiology.

ETIOLOGY (ETIOLOGICAL DIAGNOSIS)

We have already pointed out some examples in pathophysiology, therefore, here we will limit ourselves to presenting the most accepted classification at the present time, which encompasses the most frequent causes of jaundice.

A. jaundice due to increased production of free or unconjugated bilirubin that reaches the liver cell.
  1.hemolytic ulcers:
    a) Intracorpuscular: hemolytic anemia, hemoglobinopathies, enzymatic
    b) Extracorpuscular: immune agents, infectious, physical and chemical agents, hypersplenism.
    c) Excessive destruction of extravasated red blood cells: hematomas, tissue crushes, pulmonary infarctions, etc.
  2. Dyserythropoietic jaundice:
    a) Pernicious anemia.
    b) Idiopathic dyserythropoietic jaundice.
    c) Thalassemia.
    d) Erythropoietic porphyria.
  3. Short circuit hyperbilirubinemia:
    a) Pernicious anemia.
    b) Erythropoietic porphyria.
    c) In the experimental field: ligation of the common bile duct, ether, barbiturate intoxication and spinal irradiation.

B. jaundice due to disorders in the uptake of free bilirubin by the hepatocyte.
  1. Gilbert's disease.
  2. Posthepatitic hyperbilirubinemia or posthepatitic syndrome Gilbert.
  3. Drugs: bile opacifiers, flavispidic acids (active ingredient in male fern).

C. Jaundice due to disorders in the conjugation of bilirubin within the hepatocyte.
  1. Due to enzymatic immaturity:
    a) Physiological jaundice of the newborn and the premature infant.
  2. Due to absence or congenital deficiency of glucuronyl-transferase:
    a) Crigler-Najjar disease.
  3. By inhibition of glucuroconjugation:
    a) Neonatal familial hyperbilirubinemia (Lucey-Driscoll)
    b) Breast milk jaundice.
    c) Drugs: novobiocin, rifamycin.

D. jaundice due to disorders in the excretion of conjugated bilirubin from the hepatocyte to the duodenum.
  1. Intrahepatic cholestasis:
    a) Hepatocyte: Dubin-Johnson syndrome, Rotor syndrome.
    b) Canalicular: hepatitis, pregnancy, steatosis, oral contraceptives, post-necrotic cirrhosis.
    c) Cholangiolar: chlorpromazine, recurrent idiopathic cholestasis, primary biliary cirrhosis.
    d) Ductular: primary biliary cirrhosis.
    e) Septal: primary sclerosing cholangitis, cholangiocarcinoma.
  2. Extrahepatic cholestasis:
    a) Obstacles in the lumen of the bile ducts: stones (choledochal lithiasis), parasitism.
    b) Lesions of the duct wall: bile duct neoplasms, ampulloma, inflammatory or scarring strictures.
    c) Extrinsic compressions: neoplasia of the head of the pancreas, pancreatitis, lymphadenopathy, paravaterian diverticula.

ASCITIC SYNDROME

CONCEPT

Ascites is called the liquid collection located within the peritoneal cavity and in intimate contact with its two leaves, visceral and parietal. The nature of the liquid varies with the etiopathogenic factors that have intervened in its production. Normally there are 100-200 mL of fluid in the peritoneum.

There are authors who consider ascites any fluid that accumulates in the abdomen, including blood (hemoperitoneum), etc., while others only consider liquid effusions (free and transudate) as such.

More than a disease, it is a true syndrome (ascitic syndrome) common to many nosological entities.

It has been indisputably demonstrated that ascitic fluids, whatever their volume, when distributed, do not deposit exclusively in the most inclined areas, as they would in an open container, but rather adopt a special distribution that is the logical consequence of the anatomical, physical and physiological characteristics of the abdominal cavity, and not the result of chance.

The diagnosis of ascitic syndrome includes several stages:

1. Recognize its existence: positive diagnosis (syndromography)
2. Differentiate it from other conditions: differential diagnosis.
3. Specify the cause and the mechanisms that produce it: etiopathogenic diagnosis (pathophysiology and etiology).

SYNDROMOGRAPHY OR POSITIVE DIAGNOSIS

It is performed on the basis of the clinical picture and complementary examinations.

Clinical picture

For a correct clinical diagnosis we must take a thorough history. The presence of ascites should be suspected when a patient tells us that they have noticed distention or increased volume of the abdomen in the days or weeks prior to the consultation.

Sometimes some non-specific symptoms are reported, such as diffuse pain, abdominal heaviness and some functional disorders of the digestive tract, which may precede or accompany the appearance of the condition. Its appearance can be slow or abrupt; In the latter case, it is usually accompanied by major cardiorespiratory functional disorders.

At its beginning, as its installation is generally insidious, we found during the interrogation, diffuse pain in the belly, abdominal heaviness, a feeling of distension and some functional disorders of the digestive tract.

When the installation is abrupt, a rapid increase in belly volume and major functional disorders are observed.

In the state period of ascites, we have functional symptoms, physical signs and other symptoms that can also accompany them.

Functional symptoms

They can be manifest or latent, a feeling of weight in the belly or real pain; cardiorespiratory disorders with dyspnea on exertion and recumbency, tachycardia, etc.; digestive symptoms such as difficult digestion, vomiting, nausea, bloating; bladder symptoms such as dysuria, frequent urination, etc.

physical signs

According to the physical signs, ascites are divided into two large groups: free ascites, which universally and unobstructed occupies the space between both peritoneal sheets and cystic or septated ascites, in which the formation of adhesions causes the fluid to accumulate exclusively in certain sectors of the abdomen.

Free ascites, according to the amount of fluid, are divided into:

- Grade 1 ascites (or small effusion). When the volume is small, less than 3 L. Sometimes the clinical diagnosis is evident although it is not uncommon to have to perform an ultrasound for verification.

Grade 2 ascites (or medium effusion). It results when the volume of fluid ranges between 3-6 L, which makes the diagnosis evident through the physical examination of the abdomen.

- Grade 3 ascites (or large effusion). When the volume of liquid is between 6 and more than 15 L.

At least 1500 mL of fluid is needed for physical signs to appear.

Inspection. Increased abdomen in the shape of a howitzer or ostrich egg or, on the contrary, the classic batrachian belly. In abundant ascites there is deployment of the navel, the skin of the abdomen is smooth, shiny and tense, sometimes with clearly evident collateral circulation: portal, supraumbilical or periumbilical type (jellyfish head).
Possibility of umbilical hernia, exaggeration of lumbar lordosis, widening of the base of the thorax.

Palpation. Parietal tension varies with the amount of fluid and muscle tone, whether or not palpation of the abdominal viscera also depends on this. However, the belly is generally reticent. There is almost never pain, unless there is an inflammatory process. There may be a hydrocele, if the peritoneovaginal duct persists, or swelling of the labia majora, if the Nück canal persists.
Edema of the scrotum or large labia is detected only after paracentesis (subcutaneous infiltration of fluid).

Percussion. Dullness in the downward points of the abdomen that varies with changes in position. In the supine position, dullness in the flanks and above the pubis whose apparent upper limit is concave upward, with periumbilical hypersonority. In lateral recumbency, dullness on the lower flank and loudness on the upper flank; all this in the case of free spill. Percussion of the lumbar regions is dull in ascites. When the fluid is scarce, the abdomen is percussed with the patient in a standing position (hypogastric dullness). False hydrothorax of ascitic origin may appear in free forms.

In cases of septate effusions we find dull areas without postural modification alternating with dull areas: checkerboard dullness.

By tapping on one side and palpating on the opposite side, the shock of the liquid wave is perceived: transabdominal Tarral wave.

The iceberg sign appears when we make depressive, sudden and jerky movements over the epigastrium or right hypochondrium when there is hepatomegaly and over the left hypochondrium when there is gross splenomegaly. It appears as if a body (liver or spleen) with the consistency and flotation of ice, came to collide, after being submerged in the ascites fluid, against the tips of the exploring fingers.

Auscultation. It is not commonly practiced because it does not provide further diagnostic elements. Auscultation combined with percussion of two coins on the descending parts of the abdomen shows the Pitres coin sign. We can also obtain the Cruveilhier-Baunngarten sign by auscultation: murmur in the umbilical region with thrill.

Other symptoms

They vary according to the cause of ascitic syndrome.

Thus it is possible to find anemia, circulatory disorders, kidney lesions, jaundice, fever, lung lesions, opsiuria (elimination of urine several hours after meals), edema of the scrotum or large labia, hydrocele, weight loss.

Complementary exams

Radiological examinations

There are radiological signs that point to the existence of ascites and we will only list the findings of the simple abdominal examination, since the detailed description of this and other radiological methods used belongs to the subject of Radiology.

Simple radiological examination of the abdomen

It provides evidence if the following signs are observed:

Blurring of the silhouette of the intraperitoneal organs.

Erasure of the psoas line and the preperitoneal fat band.

Ground glass appearance of the intestinal loops.

Separation of the intestinal luminogram.

abdominal ultrasound

It is a useful procedure to determine the presence of fluid in the abdominal cavity. It is not only capable of detecting the presence of small liquid volumes (up to 100 mL) but also allows identifying the cause that causes it (portal hypertension, liver disease, tumors, for example).

Laboratory exams

They are performed on the ascitic fluid extracted by paracentesis, by the technique described in Chapter 62, or with the extraction by abdominal puncture with a fine needle of a small sample of the fluid (20-30 mL), which can be performed either at the midpoint of the line that goes from the navel to the anterior superior iliac spine, in the left iliac fossa, or in the middle of the umbilicopubic line.

Due to its biophysicochemical characteristics, ascitic fluid can be classified as a transudate (of mechanical origin) or as an exudate (of inflammatory origin), the differences of which are shown in Table 63.1.

Macroscopic interpretation of the liquid

The macroscopic appearance of the fluid will guide us to the possible nature of the condition.

Subject to the examination carried out by the laboratory technician, the clinician immediately notes the volume, color, viscosity and general appearance, distinguishing as main types:

1. Clear, transparent liquid, lemon yellow or greenish yellow (sallow), easy to obtain; It generally corresponds to transudates, which remind us of circulatory diseases (generalized venous hypertension), renal or hepatoportal diseases (uncomplicated liver cirrhosis). Nutritional disease should also be considered.

2. Cloudy yellow liquid. Secondary to tumors, trauma or mesenteric infarction, among others.
3. The brown liquid should always make you think of an ovarian cyst.

Table 63.4

Characteristics	Transudate	Exudate
Density	< 1015	> 1 015
Fluid protein content Fluid albumin	<30g/L	> 30g/L
content Fluid protein/Serum protein	<25g/L <	> 25g/L
Albumin gradient (serum-fluid)	0.5	> 0.5
Appearance	>1.1 gdL	< 1.1 g/dL
	Clear, transparent	Cloudy
Qualitative Rivalta *	Negative	Positive

*His technique is described in volume 1, Chapter 36 (p. 491). The quantitative Rivalta reaction and ascitic fluid electrophoresis are more accurate.

4. Hemorrhagic fluid, which suggests malignant tumors with peritoneal involvement, infarction or thrombosis, as well as some peritoneal infections, such as tuberculosis, for example.
5. Milky fluid, either chylous, which suggests processes involving the lymphatic system, as in tuberculosis, or pseudochylous, which can be seen in lues, nephritis, certain nutritional syndromes, amyloidosis, etc.
6. Of bile content. Secondary to biliary or intestinal perforations (coleperitoneum).
7. Thick, puriform liquid (similar to pus), frankly exudative, corresponds to suppurations and infections in general.
8. If the appearance is mucous and thick, it may correspond to a mucinous removal of the ovary or a pseudomyxoma of the peritoneum.
9. Gelatinous ascites, which have viscous, yellowish, stringy fluid, are observed in primitive myxoma of the peritoneum, or secondary to a neoplasia of the ovary, and in gelatinous disease of appendiceal origin.

10. Certain ascitic fluids after extraction can spontaneously clot due to excess fibrin, when they are of inflammatory origin.

Ascites cytology

When they are mechanical effusions (transudates) they contain few cellular elements represented by large isolated or crowded endothelial cells. When they are inflammatory effusions (exudates) they contain abundant cells, which can be of different types according to the causative process: lymphocytes, predominant in peritoneal tuberculosis or in attenuated banal inflammatory processes; polynuclear, predominant in acute and non-bacillary processes; large vegetating cells with nuclei in mitosis or endothelial plaques in peritoneal neoplasms; large vacuolated cells with thick nuclei and even cylindrical cells in certain ovarian cysts.

Bacteriological characteristics of ascites

The first stage to be completed is the direct examination of the liquid. For this purpose, its sediment is used, after a good centrifugation, with which smears are made and these are stained with Gram's stain or Ziehl's stain. Cultivation is also carried out.

Liquid glucose/glycemia ratio

It is important to note that the simultaneous determination of the glucose level of the ascitic fluid and serum glucose to find the mathematical relationship of their results is of great value in differentiating the diagnosis of tuberculous ascites from that of other causes of non-acute ascites, especially in places where peritoneal tuberculosis is common and laparoscopy with biopsy is not available or difficult to perform.

Sometimes, the clinical picture and the study of the ascitic fluid of liver cirrhosis is indistinguishable from that of peritoneal tuberculosis and sometimes, both entities coincide in the same patient. Furthermore, it is quite difficult to detect the tubercle bacillus in the study of the liquid. On the other hand, the macroscopic appearance of the peritoneum in laparoscopy can be confused and lead to an erroneous diagnosis of peritoneal carcinosis, if biopsies are not taken.

It has been shown that a fluid glucose/glycemia ratio of less than 0.96 is indicative of peritoneal tuberculosis, with a statistical probability of error of 0.001, while in non-acute ascites syndromes due to other causes, the ratio is above this figure and is generally greater than 1.

Due to the simplicity of the test, its cost and feasibility, it is worth systematically including it in laboratory investigations of ascitic syndrome.

Laparoscopy

It is the most important method, not only to make a positive diagnosis of ascites, but also to specify its etiology. This method is used to evacuate the ascites and perform direct observation and take biopsies, which clarify its cause.

DIFFERENTIAL DIAGNOSIS

Abdominal conditions

Ascites must be distinguished from other abdominal conditions, such

1. Adipose belly:
   a) The patient is clearly obese and the fat is distributed diffusely throughout the body.
   b) Abdominal percussion does not reveal dullness in any of the various regions of the belly. Careful examination does not allow us to complete the diagnosis.
   CO.
2. Edema of the wall:
   a) There is no liquid wave.
   b) The navel is normal.
   c) Godet exists.
3. Generalized meteorism:
   a) On percussion, hypersonority is seen throughout the abdomen, which sometimes masks hepatic dullness.
   b) Palpation of the belly does not reveal the transabdominal wave sign.
   c) Fluoroscopy shows that there is exaggerated or excessive transparency throughout the abdomen and gas collection in the loops and stomach.
4. Pneumoperitoneum. Hepatic dullness disappears.
5. Megalocolon:
   a) Usually the condition dates back to childhood and has a very chronic evolution (lasts throughout life).
   b) The disease is characterized by enormous abdominal distention and persistent constipation (for days or weeks) that alternates with diarrhea (diarrheal evacuation) that is followed by improvement in symptoms.
   c) There is often a history of intestinal obstruction crises (which sometimes lead to the death of the patient) and on radiology the colon appears of disproportionate dimensions, which is why it requires many liters of barium mixture for its complete repletion.
6. Various tumors. Hepatomegaly and splenomegaly, fibromas, bladder balloon, peritoneum tumors and hydronephrosis:
   a) There is localized dullness, delimited at their level.
   b) There is no liquid wave, there are no lateral displacements, they are
   c) The accompanying symptoms are of great value.
7. Pseudoascites. Described by Mathieu in intestinal strictures, where loops filled with fluid are deposited in the pelvis while others filled with air ascend, giving a condition that resembles ascites.

Pelvic conditions

Ascites must be distinguished from other pelvic conditions, such as:

1. Urine retention:
   a) There is a median bulge at the level of the hypogastrium (above the pubis), due to the distended bladder balloon.
   b) The upper limit of the dullness zone of the abdomen is convex upwards (it is concave in ascites).
   c) Bladder catheterization causes its evacuation, the abdominal tumor disappears, confirming the diagnosis.
2. Ovarian cyst (fig. 63.8):
   a) The dullness of the abdomen is invariable with the different postural changes of the patient and its upper limit is a curved line of superior convexity, with tympany on the flanks.
   b) The navel is normal and the belly is often asymmetrical.
   c) There are menstrual disorders and the gynecological examination shows that the tumor that represents the cyst has intimate relations with the uterus and the vaginal fornices. Let us point out, however, that a syndrome consisting of the association of an ovarian cyst or fibroma with ascites and pleural effusion (of undefined pathogenesis) has been identified, called Meigs syndrome.
3. Simple or complicated pregnancy with hydramnios:
   a) The abdominal dullness is central and has a curved upper limit, convex upwards.
   b) There are signs of pregnancy, such as amenorrhea, softened cervix, etc., but above all, the pregnancy test is positive.
   c) Ultrasound examination of the uterus verifies the existence of fetal shadow.

SYNDROMOGENESIS PATHOPHYSIOLOGY (FIG. 63.9)

There are various diseases capable of producing ascites and several factors sometimes intervene in them.

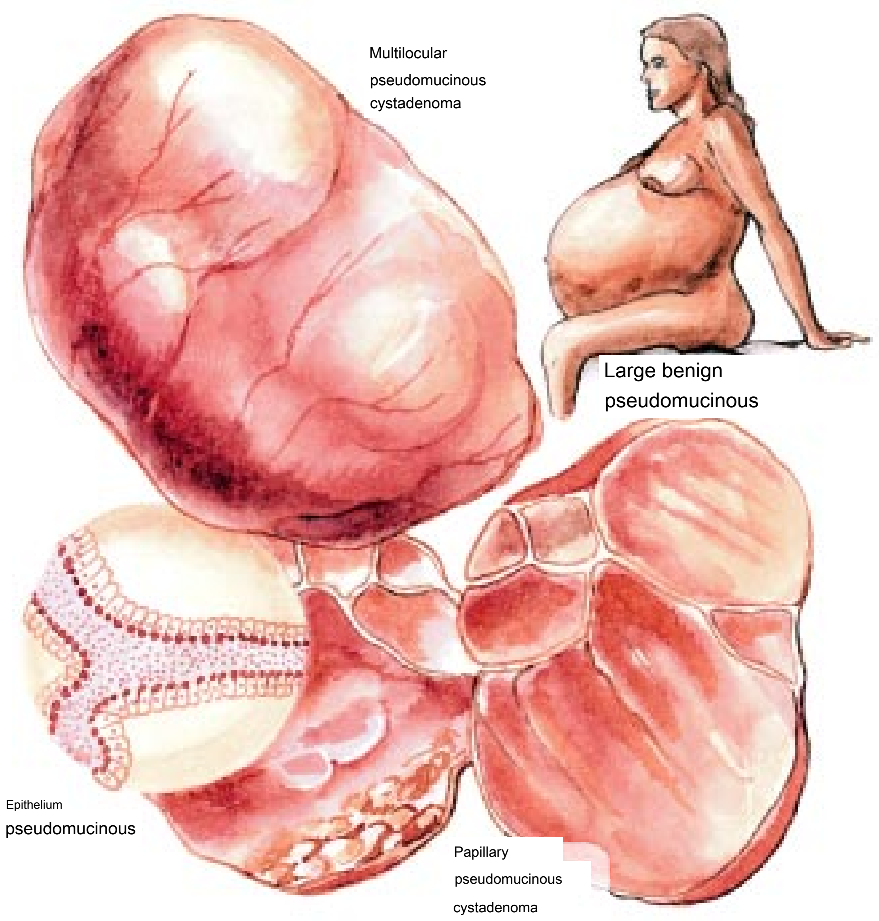

Multilocular pseudomucinous cystadenoma
Large benign pseudomucinous
Epithelium pseudomucinous
Papillary pseudomucinous cystadenoma

Fig. 63.8 Giant ovarian cyst.

its pathogenic mechanism, for which we will analyze the factors most frequently involved in the production of this syndrome:

1. Portal hypertension. Normally the portal venous pressure is 12-13 cm of water, when it exceeds 40 cm, the venous stasis in the abdominal territory opposes the normal reabsorption of the interstitial fluid. This symptom alone is not enough to cause ascites, but since it generally coexists with others, it plays its role in determining the syndrome.

2. Hypoproteinemia. When accompanied by a marked decrease in serine, it produces a decrease in the colloid osmotic pressure of the plasma.
3. Increased capillary permeability. It is caused by stasis and consecutive anoxia; It is manifested above all by the fact that in ascites, plasma proteins cross the capillary membrane (especially peritoneal) much more easily than in the normal subject. Portal hypertension, hypoproteinemia and increased capillary permeability act synergistically and exert their influence exactly at the level of the capillary barrier that separates the two compartments of the extracellular sector, which tends to disturb the normal balance of organic fluids, allowing fluid to progressively accumulate in the interstitial spaces at the expense of the plasma compartment.

4. Lymphatic hypertension. It is due to increased lymph flow due to alterations in portal circulation and liver lesions; Lymphatic obstruction by fibrin also contributes. It is a very important factor.
5. Water and sodium retention (aldosterone and antidiuretic hormone). It constitutes one of the most significant mechanisms in the production of ascites. It is caused by a decrease in glonerular filtration and, above all, by a marked increase in the tubular reabsorption of water and sodium. This last phenomenon has drawn the attention especially of researchers who have wondered what its immediate mechanism could be. Nowadays, the solution has been found and we can say that two hormones play a fundamental role in triggering hydrosaline retention by the kidney: aldosterone and the neurohypophyseal antidiuretic hormone.

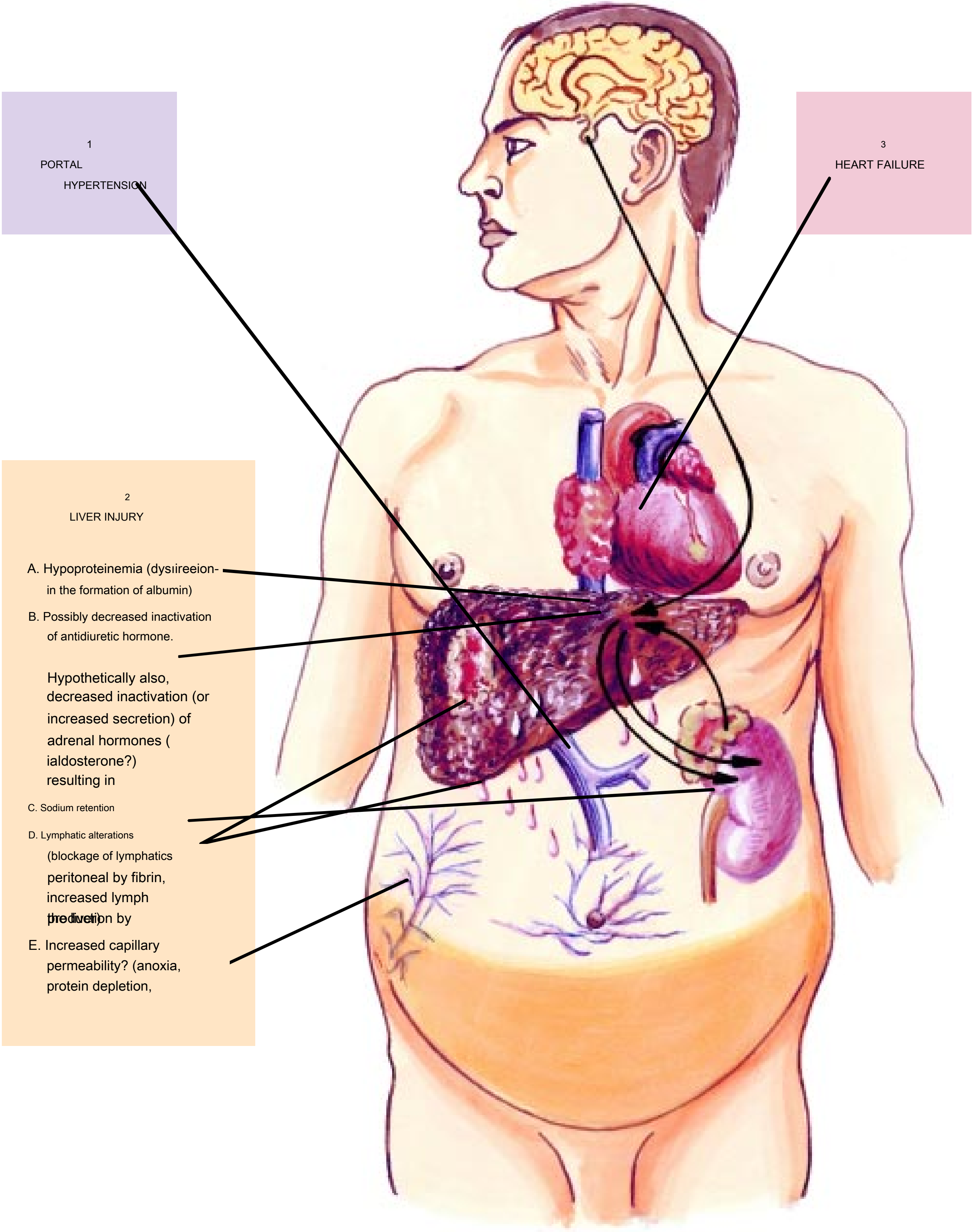

1
PORTAL
HYPERTENSION
3
HEART FAILURE
2
LIVER INJURY
A. Hypoproteinemia (dysıireeion-
in the formation of albumin)
B. Possibly decreased inactivation
of antidiuretic hormone.
Hypothetically also,
decreased inactivation (or
increased secretion) of
adrenal hormones (
ialdosterone?)
resulting in
C. Sodium retention
D. Lymphatic alterations
(blockage of lymphatics
peritoneal by fibrin,
increased lymph
production by
the liver)
E. Increased capillary
permeability? (anoxia,
protein depletion,

Fig. 63.9 Pathophysiology of ascites.

We have already studied the mechanism of action of hydrosaline retention by the kidney, when talking about edema. Let us only remember that aldosterone (the most important of the two hormones mentioned) reduces sodium excretion and increases potassium excretion through a distal tubular mechanism, thus secondarily promoting water retention. In these cases it has been proven that there is secondary hyperaldosteronism.

The neurohypophyseal antidiuretic hormone is also increased (or perhaps not sufficiently inactivated by the injured liver), stimulating water reabsorption at the distal tubule, which contributes to the production of ascites.

6. Irritation of the peritoneal serosa. This is the most important mechanism in cases of acute or chronic infections (tuberculosis) of the peritoneum, that is, in peritonitis.

Metastatic seedings in the peritoneal serosa (peritoneal carcinosis) also cause irritation of these structures and give rise to ascites that are frequently bloody.

ETIOLOGY

The diseases that can be associated with the formation of ascites are diverse and can exist alone or in combination.

1. Liver diseases:
    a) Liver cirrhosis (more than 90%).
    b) Hepatocarcinoma.
    c) Liver metastases.
2. Veno-occlusive liver diseases (mechanical ascites due to localized venous hypertension) (fig. 63.10):
    a)) Budd Chiari syndrome.
    b) Venous occlusive conditions.
3. Cardiovascular diseases (due to generalized venous hypertension) (see fig. 63.10):
    a) Constrictive pericarditis.
    b) Right heart failure.
    c) Asystoles.
    d) Chronic cor pulmonale.
4. Pancreatic diseases:
    a) Acute pancreatitis.
    b) Pancreatic neoplasia.
5. Kidney diseases:
    a) Nephritis.
    b) Renal arteriosclerosis.
    c) Polycystic kidneys.
6. Malignant diseases of the abdomen:
7. Nutritional deficiencies.
8. Conditions of the peritoneum: TB peritonitis, carcinomatosis, rheumatic peritonitis.
9. Obstruction of lymphatic vessels or thoracic duct.
10. Gynecological diseases: uterine or ovarian tumors.
11. Myxedema.

CHRONIC DIARRHEAL SYNDROMES

There are multiple symptoms of chronic diarrhea caused by innumerable causes, both functional and organic; Thus we have chronic irritable bowel diarrhea, colonic diverticulosis, so-called allergic diarrhea, etc.

But under the term of chronic diarrheal syndromes we are going to consider two groups that truly constitute syndromes, since in them there is not only chronic diarrhea, but a constellation of symptoms and signs that give them their own personality, and that on the other hand obey different pathophysiological mechanisms. These two syndromes are:

Malabsorption syndrome.

Dysenteric syndrome.

MALABSORPTION SYNDROME

Concept

The term malabsorption is strictly used to designate defective absorption of any nutrient, which leads to increased fecal losses.

Conventionally, it is applied when there is defective absorption of fats, proteins, carbohydrates and vitamins, the clinical manifestations of which are:
high-type diarrhea with or without steatorrhea, anemia, weight loss, weakness and symptoms of vitamin deficiency, the most frequent of which is glossitis.

This term must be differentiated from maldigestion that results from an increase in fecal losses of nutritional substances due to defective digestion, both at the gastric and biliopancreatic levels.

Although both processes have different pathophysiology, in practice it is difficult to discern them clinically, due to the great similarity of their symptoms.

Tropical sprue and celiac disease constitute the most representative nosological entities of this syndrome.

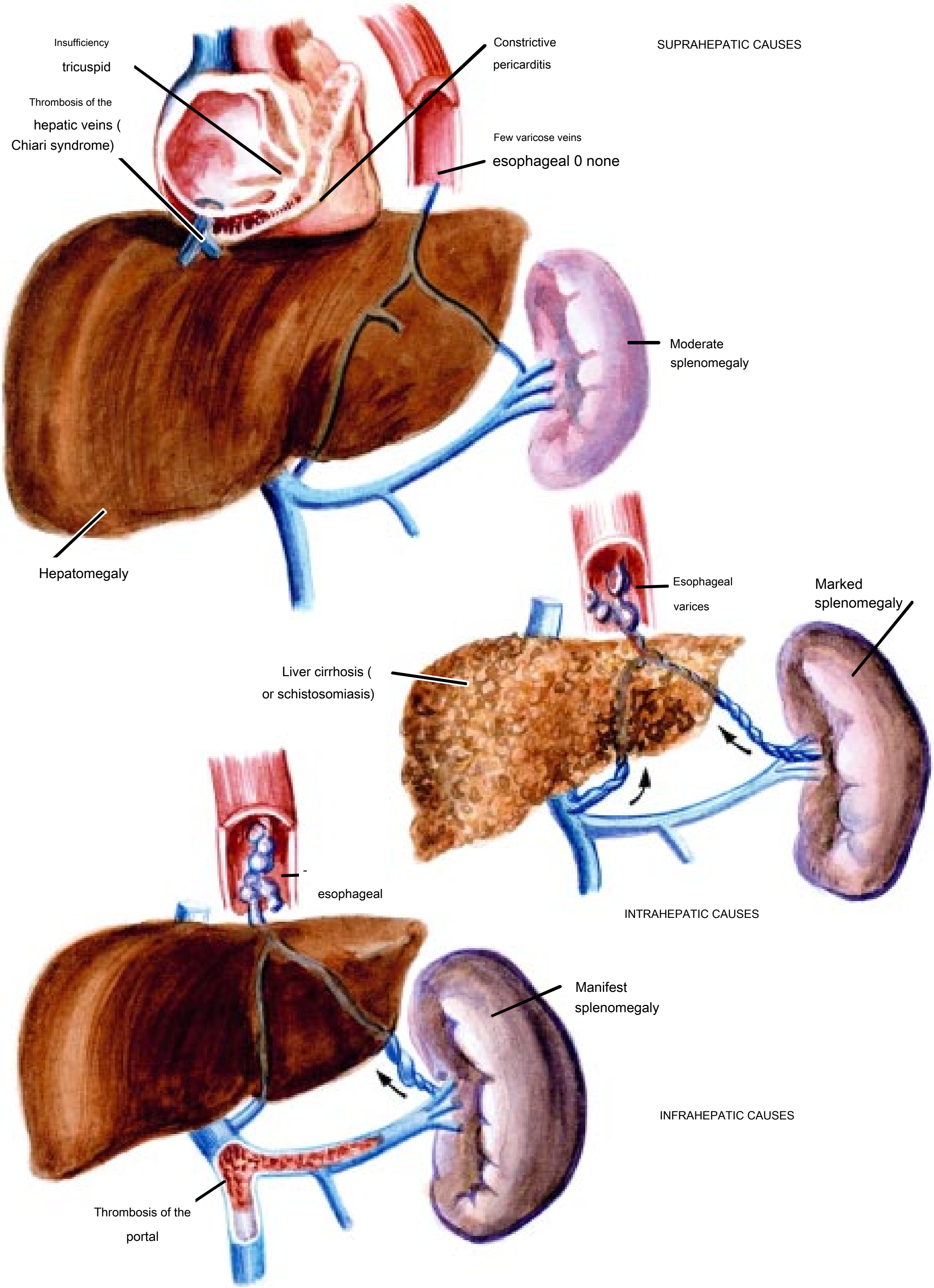

Insufficiency
tricuspid
Thrombosis of the
hepatic veins (
Chiari syndrome)
Constrictive
pericarditis
SUPRAHEPATIC CAUSES
Few varicose veins
esophageal 0 none
Moderate
splenomegaly
Hepatomegaly
Esophageal
varices
Marked
splenomegaly
Liver cirrhosis (
or schistosomiasis)
esophageal
INTRAHEPATIC CAUSES
Manifest
splenomegaly
INFRAHEPATIC CAUSES
Thrombosis of the
portal

Fig. 63.10 Ascites of mechanical cause.

Classification

Given the diversity of etiological and pathogenic factors that determine this syndrome, it is necessary to try to classify it for a better understanding. Below we offer a simple and practically useful classification:

- Primary malabsorption syndromes.
- Secondary malabsorption syndromes.

Primary malabsorption syndromes

Those malabsorption syndromes that depend on a primitive or primary disorder of the intestinal mucosa are considered primary.

Tropical sprue, celiac disease and primary hypolactasia belong to this group.

Secondary malabsorption syndromes 0 symptomatic

Under this title are grouped all those malabsorption syndromes whose production mechanism involves several causes. In turn, they are subdivided into two large groups: those secondary to digestive insufficiency and those secondary to an absorption disorder.

As an example of digestive insufficiency we have secondary malabsorption due to pancreatic insufficiency, hepatic insufficiency and gastric insufficiency. And as an example of absorption disorders we have secondary malabsorption due to giardiasis, intestinal tuberculosis, Crohn's disease of the small intestine, etc.

Syndromogenesis or pathophysiology

There are multiple pathophysiological mechanisms that produce a malabsorption syndrome; Below we list the fundamental ones:

Deficiency of cofactors necessary for the preparation of the nutrients that are going to be absorbed.

-Shortening of the contact time of nutrients with the mucosa.

- Decrease in intestinal absorption surface.

Decrease in the digestive enzymes of the intestinal mucosa.

- Disorders of specific transport mechanisms.
- Disorders of vascular outflow from the intestine.
- Presence of abnormal flora.

Deficiency of cofactors necessary for the preparation of the nutrients that are going to be absorbed

The lack of cofactors necessary to prepare nutrients so that they can be absorbed is due to the mechanisms accompanying diseases of the stomach, pancreas and hepatobiliary system.

Pancreatic insufficiency causes generalized malabsorption of carbohydrates, proteins and lipids, including, of course, fat-soluble vitamins, since there is a deficiency of proteolytic, aminolytic and lipolytic enzymes.

Gastric insufficiency is another example of these alterations, where a deficiency of intrinsic factor and hydrogen ions may occur. The deficiency of intrinsic factor (IF) causes malabsorption of vitamin B. On the other hand, calcium and iron, which require an acidic pH for absorption, will be affected in the presence of gastric insufficiency (hydrogen ion deficiency).

In the course of liver disease and biliary obstructions there is a deficiency of bile salts, which are essential for the micellar solubilization of fats, the way in which these nutrients are absorbed by the intestinal mucosa.

Shortening the contact time of nutrients with the mucosa

When intestinal transit becomes abnormally accelerated, poor absorption of various nutrients occurs due to the short contact time between them and the intestinal mucosa; This is one of the mechanisms that has been considered in the malabsorption of gastrectomized and hyperthyroid patients.

Decreased intestinal absorption surface

The decrease in the absorption surface explains the absorptive disorders after intestinal resections, surgical diversions, fistulas, etc., therefore the intensity of malabsorption depends on the location and extent of the resected segment.

Decrease in the digestive enzymes of the intestinal mucosa

The deficiency of enzymes specific to the intestinal mucosa finds its best examples in celiac disease, where there is a deficiency of enzymes to digest gluten; Among the enzymatic deficits, one of the most frequent in daily practice is that of lactase disaccharidase, an enzyme that splits lactose (milk sugar) into glucose and galactose. Since this enzyme does not exist, patients with this deficiency do not tolerate milk or its derivatives, since lactose, not being hydrolyzed by lactase, causes an increase in osmotic pressure, which results in an accumulation of fluids within the intestinal lumen.

Disorders of specific transport mechanisms

These disorders cause malabsorption only in those nutrients that require a specific mechanism. These are generally inherited defects. An example of this mechanism is the absence of beta lipoprotein, which causes an inability for lipids to form chylomicrons, which prevents them from passing through the cell.

thing to the intestinal lymphatics.

Another example of this mechanism is vitamin B malabsorption, due to the absence of receptors in the ileum.

Disorders of vascular outflow from the intestine

The typical example of these disorders is the obstruction of regional lymphatics due to inflammatory processes or neoplastic processes.

Presence of abnormal flora

Bacterial proliferation in the small intestine is observed in different conditions that favor stasis: jejunal diverticulosis, strictures and fixed loops; The latter can be produced by mechanical causes (adhesions) or by hypomotility of various origins such as diabetes (neuropathy) and scleroderma (wall infiltration), among other conditions.

The alteration of the metabolism of bile salts by bacteria explains the pathophysiology of this disorder.

Bacteria deconjugate bile salts; These deconjugated salts lack the optimal properties for micellar formation, which is an essential condition for the absorption of lipids, and as a consequence of this alteration, steatorrhea appears.

Syndromography or positive diagnosis

The fundamental symptoms and signs have been expressed in the concept of this syndrome, but we can add that the onset is generally insidious, although some diseases may have an acute onset, as is the case of tropical sprue.

Clinical picture

It is mainly caused by: liquid, pasty or semi-liquid diarrhea, abundant, with food remains, sometimes greasy, that leaves anal burning; asthenia, marked anorexia, weight loss, mouth ulcers, glossitis, skin spots, anemia and hypoproteinemia, hemorrhagic, bone and neurological manifestations, in very advanced SOS cases.

To this general clinical picture that expresses the absorptive deficit, the clinical pictures of the diseases that cause it will be added.

It is important to note that there are asymptomatic forms, where only alterations are found in laboratory tests, and florid polysymptomatic forms.

Complementary exams

Biochemical studies can reveal a disorder of intestinal absorption, both by indirect and direct methods.

The indirect demonstration of an absorption disorder can be manifested by the deficiency of a substrate in the body tissues; But this implies a very long period of malabsorption, which is why these methods are of little use in the initial stages. An example is the dosage of carotenes in the blood.

A direct method is one that carries out tolerance tests by orally administering the substrate, calculating the amount absorbed by the assessment of fecal residue or by blood and urinary values.

Lipid absorption

Van de Kamer: This method of analysis of fecal lipids is the most used to demonstrate the presence of steatorrhea. By this method, a normal subject with a diet of 50-150 g of fat should have a fecal fat of 5g or less in 24 hours. Figures greater than 5 g are considered pathological.

Other methods that measure fat absorption are those that use radioactive isotopes such as triolein I'sl and oleic acid I'31. The vitamin A tolerance test is another study, but rarely used.

carbohydrate absorption

Caused hyperglycemia. A flat curve is obtained in malabsorption syndromes, but it is an insensitive indicator, since the glucose transport capacity is so great that a very diffuse disease would be needed to obtain abnormal tests. On the other hand, a flat curve may be the expression of rapid utilization and not malabsorption.

D-xylose. This substance is a pentose that cannot be transported by the normal hexose mechanism, and is partially metabolized in the body. It is the simplest and simplest test to assess the function of the proximal intestine. Normal subjects will eliminate 20% of the ingested D-xylose in the urine, and patients with absorption disorders will eliminate figures less than 20%.

To evaluate this test, it is necessary to rule out the existence of kidney failure, since in these cases it gives false positive results.

Lactose tolerance test (TTL). It allows the assessment of intestinal lactase deficiencies, since this test is closely related to the activity of this enzyme. Increases in blood glucose above 20 mL above fasting levels are considered normal figures.

Increases less than 20 mL are considered pathological. In lactase deficiencies, a flat blood glucose curve is observed during the test and symptoms such as gas, abdominal cramps and diarrhea appear, which are of great diagnostic value. Some factors such as delayed gastric emptying can give false positive results.

protein absorption

Marked albumen. Albumin labeled with Crsl or I31 is used. It is administered orally and it is determined in feces, blood or urine by measuring radioactivity. Normal figures are considered when there is less than 5% of the ingested radioactivity in the feces.

Fecal nitrogen. Its determination is made by the Kjeldahl method, whose normal values are 2.2-2.5 g in 24 hours, for an average intake of 100-120 g of protein daily. Figures greater than 3 g indicate manifest crea- tower.

Jejunum biopsy

The histological study of the jejunal mucosa allows in many cases to make the etiological diagnosis of this syndrome.

Intestinal villous atrophy can be partial or total; This lesion is characteristic of tropical sprue and celiac disease, although it can be seen in cases of intestinal parasitism, malnutrition and others.

In digestive insufficiencies, the jejunum biopsy is normal, as well as in primary hypolactasias and bacterial overgrowths.

Histology of the jejunum will also provide specific data according to the etiological cause of the syndrome (lymphoma, Whipple's disease, scleroderma, etc.).

radiological studies

Intestinal transit offers data of great interest in the study of this syndrome, which can show everything from a pattern similar to that of sprue (flocculation, dispersion and segmentation of barium, etc.), to images of stenosis and dilations as can be observed in regional ileitis, intestinal tuberculosis, lymphomas, etc.

Etiology

We are going to consider the most frequent etiologies of this syndrome, following the order we gave when classifying them:

A. Primary malabsorption syndromes.
   1. Tropical sprue.
   2. Celiac disease.
   3. Primary hypolactasia.

B. Secondary malabsorption syndromes.
   1. Due to digestive failure:
      a) Pancreatic insufficiency (enzyme deficiency): chronic pancreatitis, pancreatic fistulas and cystic fibrosis of the pancreas.
      b) Liver failure (lack of bile salts): cirrhosis, hepatitis and bile duct obstruction.
      c) Gastric insufficiency (lack of hydrochloric acid): gastric aquilia, pyloroplasty and gastrectomy.
   2. Due to absorption disorders:
      a) Inflammatory disease of the small intestine: regional enteritis, tuberculosis, nonspecific granulomatosis and intestinal
      b) Lymphatic obstruction: lymphomas, Whipple's disease and carcinoma.
      c) Systemic disease: amyloidosis, scleroderma and carcinoma.
      d) Resections of the small intestine.
      e) Endocrinopathies.
      f) Vascular insufficiency.
      g) Postantibiotics and cytostatics.
      h) Zollinger-Ellison syndrome.
      i) Bacterial overgrowth.
      j) Starvation.

DYSENTERIC SYNDROME

Concept

We know as dysenteric syndrome the set of symptoms and signs that express the reaction of the rectal mucosa to different lesions: inflammatory, parasitic or tumorous.

The triad that characterizes this syndrome is given by:

1. Abnormal stools with mucus, pus and blood.
2. Pushing.
3. We have.

The typical deposition of this syndrome is afecal, reminiscent of sputum, however, it can be accompanied of molded, pasty or liquid stools. The presence of this syndrome does not mean that the rectum is always injured exclusively, as it may be a diffuse process that affects the entire colon and also the rectum.

Syndromogenesis or pathophysiology

There are multiple mechanisms involved in the production of this syndrome, which can be grouped into: general and specific mechanisms.

General mechanisms

Alterations of the intestinal mucosa and neuromotor balance are the general mechanisms that explain this syndrome, regardless of the etiological agent.

Intestinal mucosal alterations

When the intestinal mucosa becomes inflamed, it incorporates the products derived from this injury into the excrement, that is, mucus, blood and epithelium, characteristic elements of abnormal deposition. These elements fluidize the deposition and accelerate intestinal transit, causing what is known as organic diarrhea.

Neuromotor balance alterations

This mechanism refers to the alteration of different neural factors. Among them: hypermotility, exaggeration of gastroileal and gastrocolic reflexes, and alterations nes of the central nervous system.

It has been shown that in patients with diarrhea, increased defecation is accompanied by rhythmic and vigorous activity of the right colon, while the sigmoid region presents longitudinal contraction with shortening, without rhythmic contractions. This sigmoid hypomotility is an important part of the diarrhea mechanism.

Specific mechanisms

They depend on the causative agent and vary according to the pathogenic action of each etiological agent. We will point out the most frequent ones:

Shigellosis

The toxins produced by Shigella bacilli cause acute inflammation of the colon, with coagulation necrosis and ulcerations.

In general, the rectosigmoid region is the most frequent and intensely affected.

It is assumed that the toxicity is due to the somatic antigen, which would act on the small blood vessels and could cause hemorrhage.

It is considered that the invasion of the mucosa by the bacillus causes alterations in the flow and transport of electrolytes.

Salmonellosis

This infection is rather generalized, and not localized to the rectum, since it invades the thoracic duct through the intestinal lymph nodes and later the general circulation (septicemic phase), which produces lesions in various organs and systems.

Idiopathic ulcerative colitis

In this entity characterized by bloody diarrhea, with straining and tenesmus, the causative agent has not yet been determined, although there is evidence that allows it to be classified as an autoimmune disease. The most recent studies indicate that sensitized lymphocytes are responsible for mucosal damage.

These lesions are caused by congestion, edema and chronic inflammatory infiltrate of the rectocolonic mucosa, with ulcerations and marked friability, which easily explains the clinical picture of these patients.

Anebic colitis and schistosomiasis

In these parasitosis, the mechanism of production of the syndrome is obvious, that is, it is produced by the action of the parasite on the colonic mucosa. Amoebic dysentery is due to the development, deep in the colonic mucosa, of the histolytic amoeba, that is, the trophozoite; while in schistosomiasis the lesion is caused by the embedding of the eggs of Schistosoma species under the intestinal mucosa. Such parasites produce ulcerations of the mucosa, which explains the symptoms.

Malignant and benign tumors of the rectum

Invasion of the rectum or sigmoid by a malignant lesion is one of the most frequent causes of dysenteric syndromes in our environment. The pathogenesis in these cases is due to the fact that the neoplastic tissue ulcerates easily and incorporates the products of its disintegration into the deposition.

Regarding benign tumors, the most common is the adenomatous polyp, which can cause bleeding when ulcerated, as well as increased mucous secretion.

The villous tumor, with great malignant potential, can produce abnormal mucous-type secretions through the rectum; Frequently this information is the only one offered by the patient.

Syndromography or positive diagnosis

clinical picture

As we noted in the definition, the clinical triad that individualizes this syndrome is given by abnormal stools (mucopyousanguinous), straining, and tenesmus.

But there are other symptoms and signs that depend on the causing disease. The mode of onset can be abrupt, with alterations in the hydromineral balance and a major impact on the general state, as seen in bacterial dysenteries, or it can be insidious in nature, as in tumor lesions.

In neoplastic processes, anemia, weight loss, asthenia and anorexia are also observed, according to the degree of invasion of the tumor process, which may be accompanied by other manifestations that depend on the metastatic locations (for example, nodular hepatomegaly).

In inflammatory diseases of the large intestine (ulcerative and granulomatous colitis), in addition to the dysenteric syndrome, systemic manifestations can be observed (arthralgias, dermatopathies, liver diseases and others).

Bacterial processes are generally characterized by acute onset with septic manifestations (fever, leukocytosis, stuporous state).

From the above, it follows that this syndrome does not allow, just because it is present, an etiological diagnosis to be made, since it is a common denominator of multiple diseases; but its presence guides and forces the doctor to rule out rectal, rectocolonic or neighboring pathologies.

As part of the physical examination, a careful inspection of the margins of the anus is required, as well as a rectal examination, which can often lead to suspicion of the cause of the syndrome.

Complementary exams

The basic complementary tests for the diagnosis of this syndrome are:

1. Rectosigmoidoscopy with biopsy.
2. Coproculture.
3. Parasitological examination of feces.
4. Radiological study of the colon.

These studies will allow us to elucidate the etiology of this syndrome.

Rectosigmoidoscopy not only allows us to identify the state of the mucosa, whether it is ulcerated, congested or if there is a tumor, but through it samples can be obtained for histological study and mucosal scrapings to search for parasites.

It should be noted that in very acute states this exploration must be carried out by very experienced hands, since there is a danger of perforation.

Fecal cultures, in search of bacterial agents, and serial parasitological examinations are also of great help in the etiological diagnosis.

The radiological study of the colon allows us to clarify whether it is a diffuse condition of the colon or located in the rectosigmoid.

Colonoscopy is decisive when other examinations have not defined the diagnosis.

Etiology

For your better understanding, we are going to group the causes of the syndrome into four large groups: inflammatory, parasitic, tumorous and others.

1. Dysenteric syndrome of inflammatory origin:
   - a) Idiopathic ulcerative colitis.
   - b) Granulomatous colitis or Crohn's disease of rectocolonic location.
   - c) Tuberculosis of rectocolonic location.
   - d) Post-radiation proctitis.
   - e) Nicolas and Favre disease.
   - f) Rectitis due to abuse of suppositories.
   - g) Bacillary dysentery.
   - h) Nonspecific rectocolitis.
2. Dysenteric syndrome of parasitic origin:
   - a) Amoebic colitis.
   - b) Schistosomiasis colitis.
   - c) Balantidiasis.
3. Dysenteric syndrome of tumor origin:
   - a) Malignant tumors of the rectosigmoid.
   - b) Benign tumors of the rectosigmoid.
4. Dysenteric syndrome of various origins:
   - a) Rectal location of hemopathies (lymphoid leukosis, for example).
   - b) Hemorrhoids, papillitis and cryptitis of the anus.
   - c) Anal fissures.
   - d) Neighborhood processes: infections of the lower pelvis that irritate the rectum due to contiguity, malignant neoformations of bladder or gynecological origin that invade the rectal wall, etc.
   - e) Allergic and post-antibiotic rectocolitis.

DIGESTIVE BLEEDING

CONCEPT

This syndrome basically consists of the expulsion of blood from a lesion located in the digestive tract, from the mouth to the anus. It has two fundamental ways of manifesting itself: either as the expulsion of red or black blood from the mouth in the form of vomiting (hematemesis), or from the rectum in the form of blackish stools (melena) or red (enterorrhagia).

which depends on the location of the hemorrhage, but above all on its massiveness and other associated factors, as has been pointed out in the study of the symptoms hematemesis, melena and enterorrhagia.

SYNDROMOGENESIS OR PHYSIOPATHOLOGY

The fundamental production mechanisms of bleeding are injuries to the arteries, veins and capillaries, which allow blood to escape from the vessels in greater or lesser quantities.

As we said in the hematemesis section, the most common vascular lesions are: rupture, thrombosis, embolism, exulcerations, as well as parietal lesions of the vessels and vascular neoformations.

Other possible mechanisms are changes in intravascular pressure and alterations in the factors involved in blood clotting. These factors have been explained when studying hematemesis.

SYNDROMOGRAPHY 0 POSITIVE DIAGNOSIS

As we explained in the study of hematemesis, the differential diagnosis must be made with blood coming from other organs not dependent on the digestive tract, such as:

Epistaxis.
Hemoptysis.
- Stomatorrhagia.

Epistaxis. Hemorrhage that comes from the nasal passages and can be expelled through the mouth.

Hemoptysis. Respiratory symptom that consists of the expulsion of foamy, sparkling blood, accompanied by other respiratory symptoms.

Stomatorrhagia. Blood produced in the oral cavity itself, in which case, a quick physical examination of the mouth can verify it.

The diagnosis of hematemesis of melena is usually easy, but in cases of scant bleeding, complementary methods (determination of occult blood) must be used to diagnose this syndrome.

During its passage through the intestine the blood becomes darker and sometimes black. This alteration in blood color depends on several factors such as: location of bleeding, amount of bleeding and speed of intestinal transit.

The usual origin of the melena is in the first part of the duodenum or in the proximal regions of the jejunum, although sometimes hemorrhages from the lower portion of the small intestine or the right colon are also accompanied by blackish stools and not red blood.

If intestinal transit is very rapid, some of the blood in the stool will be red, as occurs after heavy bleeding from a duodenal ulcer.

It is estimated that the presence of only about 60 mL of free blood in the upper digestive tract is enough to cause a melena.

Clinical picture

The clinical picture will depend on the amount of blood expelled, thus dizziness, vertigo, fatigue, skin-mucous pallor, tachycardia and even shock may be seen in massive bleeding. Tachycardia and pallor are signs of great value in making the diagnosis of hemorrhage even before it manifests itself.

Epigastralgia and gastric burning are symptoms that usually accompany gastric and duodenal ulcers.

The obvious physical signs of liver cirrhosis, such as hepatomegaly, splenomegaly, ascites, telangiectasias, will help to consider the diagnosis of ruptured esophageal varices.

Hemorrhagic manifestations (petechiae, ecchymoses) on the skin should suggest blood dyscrasias with alterations in coagulation factors.

A history of intestinal cramps, constipation alternating with diarrhea, and rectal tenesmus will lead one to think of ileal or colorectal lesions.

Complementary exams

Today, the most effective complementary method for diagnosing upper digestive bleeding is digestive endoscopy (esophagogastroduodenoscopy), which can be performed urgently if the patient's physical condition allows it. In low bleeding, rectosigmoidoscopy or colonoscopy can be used.

Contrasted radiological studies of the esophagus, stomach, duodenum, jejunum, ileum and colon are of little use in the acute phase, but they can be used in the diagnosis of ulcers, diverticula, esophageal varices, polyps or tumors when an endoscopy service is not available.

Another radiological study that is also useful is selective arteriography of the superior or inferior mesenteric artery, which sometimes makes it possible to locate the site of the bleeding vessel. This method is best indicated in massive hemorrhages that prevent endoscopy and in jejunoileal lesions.

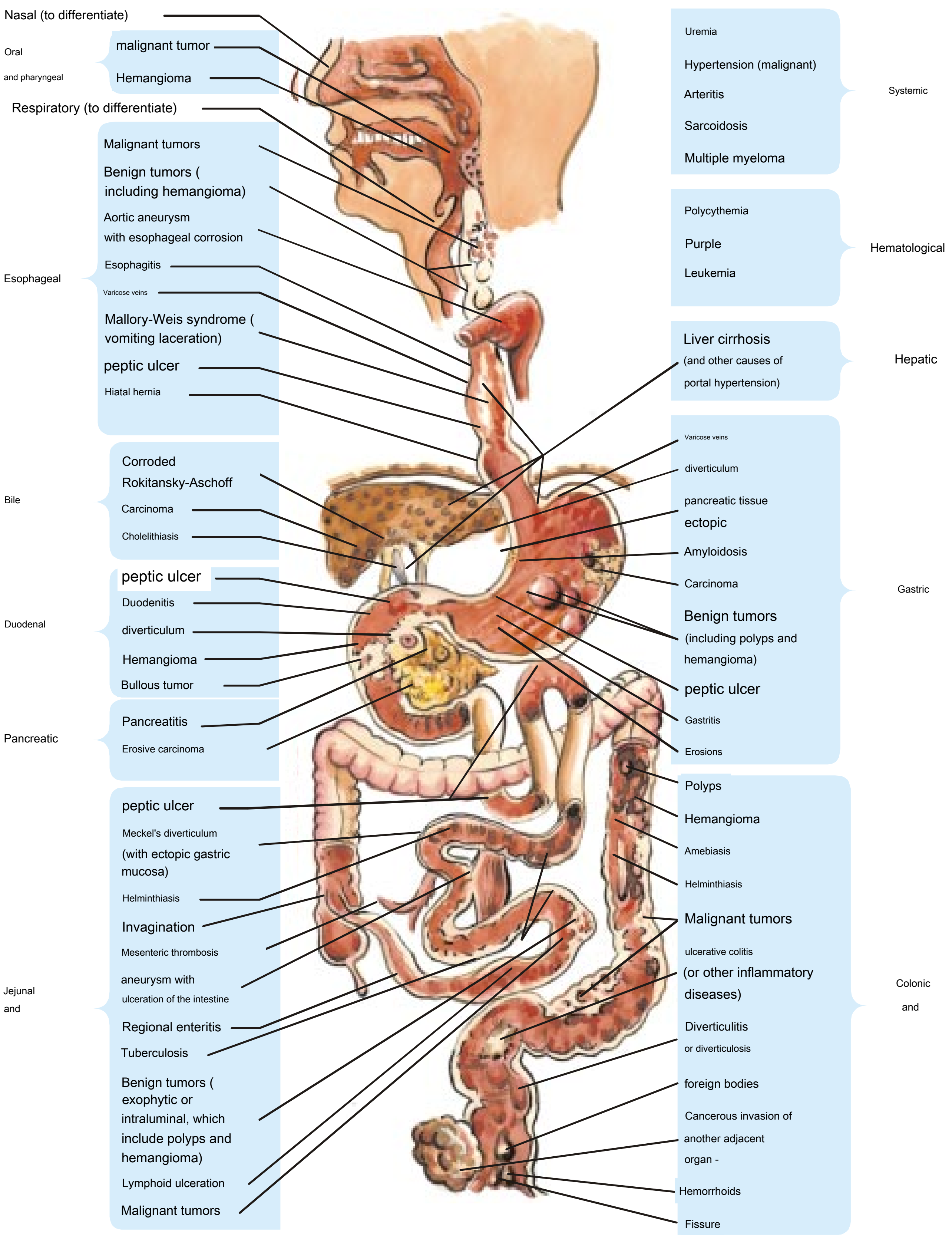

Nasal (to differentiate)
Oral
and pharyngeal
malignant tumor
Hemangioma
Respiratory (to differentiate)
Esophageal
Malignant tumors
Benign tumors (including hemangioma)
Aortic aneurysm with esophageal corrosion
Esophagitis
Varicose veins
Mallory-Weis syndrome (vomiting laceration)
peptic ulcer
Hiatal hernia
Bile
Corroded
Rokitansky-Aschoff
Carcinoma
Cholelithiasis
Duodenal
peptic ulcer
Duodenitis
diverticulum
Hemangioma
Bullous tumor
Pancreatic
Pancreatitis
Erosive carcinoma
Jejunal
and
peptic ulcer
Meckel's diverticulum (with ectopic gastric mucosa)
Helminthiasis
Invagination
Mesenteric thrombosis
aneurysm with ulceration of the intestine
Regional enteritis
Tuberculosis
Benign tumors (exophytic or intraluminal, which include polyps and hemangioma)
Lymphoid ulceration
Malignant tumors
Uremia
Hypertension (malignant)
Arteritis
Sarcoidosis
Multiple myeloma
Systemic
Polycythemia
Purple
Leukemia
Hematological
Liver cirrhosis
(and other causes of portal hypertension)
Hepatic
Varicose veins
diverticulum
pancreatic tissue
ectopic
Amyloidosis
Carcinoma
Benign tumors
(including polyps and hemangioma)
peptic ulcer
Gastritis
Erosions
Gastric
Polyps
Hemangioma
Amebiasis
Helminthiasis
Malignant tumors
ulcerative colitis
(or other inflammatory diseases)
Diverticulitis
or diverticulosis
foreign bodies
Cancerous invasion of another adjacent organ -
Hemorrhoids
Fissure
Colonic
and

Fig. 63.11 Etiology of digestive bleeding.

ETIOLOGY (FIG. 63.11)

Let's point out the most frequent causes that cause digestive bleeding at different levels:

1. Diseases of the mouth and pharynx. Ulcers and tumors of the mouth, pharynx and tonsils, trauma and streptococcal infections, gingivitis, varicose veins at the base of the tongue, etc.
2. Diseases of the esophagus. Esophageal varices, esophagitis, esophageal ulcers, benign and malignant tumors of the esophagus, diverticula, Mallory-Weiss syndrome, etc.
3. Diseases of the stomach and duodenum. Gastric and duodenal ulcers, acute and chronic gastritis, erosive gastritis, benign and malignant tumors, diaphragmatic hiatus hernia, trauma, foreign bodies, prolapse of the gastric mucosa in the duodenum, diverticula, rupture of a sclerotic vessel, acute duodenitis, etc.
4. Diseases of the bile ducts (hemobilia). Hemorrhage within the biliary tract is called hemobilia. This can be caused by intrahepatic or extrahepatic diseases. Among intrahepatic diseases, the most common cause is non-penetrating hepatic trauma, which results in the formation of a hematoma, which drains into the biliary tree. Among the extrahepatic causes, the most common is hepatic artery aneurysm. Neoplasia of the ampulla of Vater (ampulloma) can cause digestive bleeding.
5. Diseases of the small intestine. Benign tumors, including hemangiomas, frequently cause bleeding that is difficult to diagnose, ulcers of the jejunum or ileum, Meckel's diverticulum, regional enteritis, acute hemorrhagic enterocolitis, mesenteric thrombosis, as well as volvulus and intestinal intussusception.
6. Diseases of the colon and rectum. Benign tumors (polyps, hemangiomas), malignant tumors, diverticulitis or diverticulosis, ulcerative colitis, parasitic conditions (amoebas), hemorrhoids, fissures and foreign bodies in the rectum.
7. Diseases of the blood (haemopathy) and blood vessels. Many blood dyscrasias can sometimes be accompanied by digestive bleeding. Among them we will mention the following: polycythemia vera, leukemias and lymphomas, thrombocytopenic purpura, hemophilia, etc.

   Among blood vessel diseases, a rare cause of digestive bleeding is hereditary hemorrhagic telangiectasia (Rendu-Osler disease).

8. Other diseases. Other conditions capable of producing digestive bleeding at a given time in their evolution are: amyloidosis, multiple myeloma, uremia, shock, etc.

64

HEMOLYMPHOPOIETIC SYSTEM. HEMATOPOIESIS AND MAIN SYMPTOMS AND SIGNS

HEMA TOPOIESIS

ORGANIZATION OF HEMATOPOIETIC CELLS

Three main categories of mature cells appear in peripheral blood: red blood cells (erythrocytes), thrombocytes (platelets) and white series cells that group lymphocytes, monocytes, neutrophils, eosinophils and mastoid cells (mast cells or basophils). Each of these mature cells is specialized in carrying out a precise function (defense against infection, oxygen transport, primary hemostasis, etc.). To maintain a constant number, they need to be replaced continually throughout their lives and this replacement depends on the proliferation and differentiation of the progenitor cells. The process of forming blood cells is called hematopoiesis (Fig. 64.1). In humans, the bone marrow is the main hematopoietic organ.

COMPARTIMENTAL MODEL OF HEMATOPOIESIS

Currently, the compartmental model is divided into four compartments, each of which corresponds to a specific stage of differentiation (fig. 64.2). The first compartment, the most primitive, consists of totipotent and multi- or pluripotent stem cells. These are characterized by their ability to give rise to identical stem cells (self-renewal); A very limited proportion of these cells is proliferating, but this capacity for proliferation is not very extensive. Actually, the proliferation and differentiation of a pluripotent stem cell can give rise to about 10 committed progenitor cells (committed stem cells), which are programmed to differentiate into one, or eventually two, of the three specific lines (erythroid, granule-nmacrophagocytic and megakaryocytic). These committed progenitor cells, which represent the second compartment, have very little or almost no capacity for self-renewal, but they have an important capacity for proliferation. They give rise to morphologically recognizable blast cells (third compartment), capable of limited proliferation.

Finally, the fourth compartment consists of differentiated and functional mature cells, without the ability to proliferate and with a limited lifespan, such as erythrocytes, granulocytes and thrombocytes.

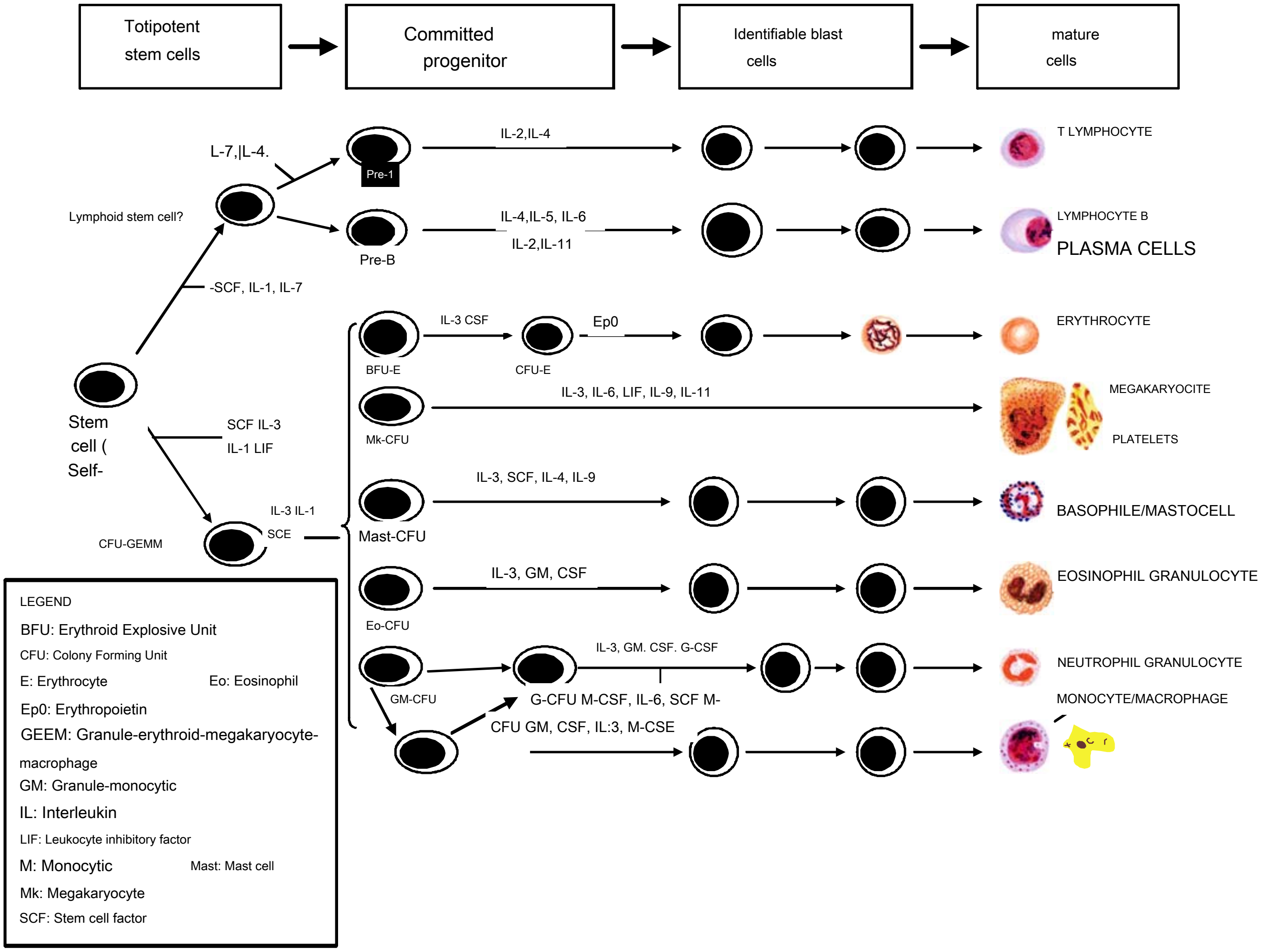
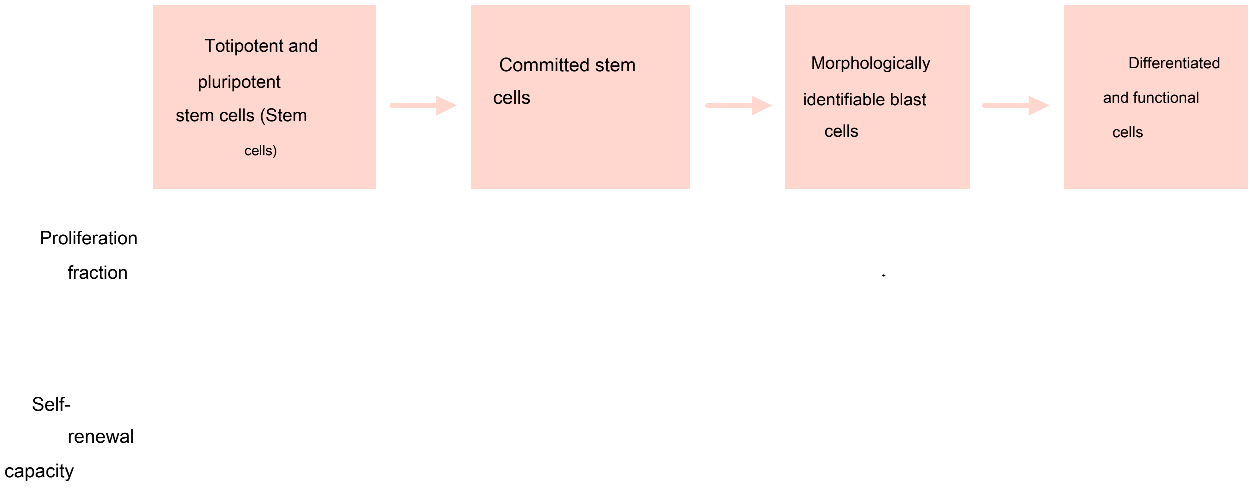

Totipotent stem cells
Committed progenitor
Identifiable blast cells
mature cells
L-7,|L-4.
Pre-1
IL-2,IL-4
T LYMPHOCYTE
Lymphoid stem cell?
IL-4,IL-5, IL-6
IL-2,IL-11
Pre-B
LYMPHOCYTE B
PLASMA CELLS
-SCF, IL-1, IL-7
IL-3 CSF
Ep0
BFU-E
CFU-E
ERYTHROCYTE
IL-3, IL-6, LIF, IL-9, IL-11
Mk-CFU
MEGAKARYOCITE
PLATELETS
Stem cell (Self-
SCF IL-3
IL-1 LIF
IL-3, SCF, IL-4, IL-9
IL-3 IL-1
SCE
CFU-GEMM
Mast-CFU
BASOPHILE/MASTOCELL
IL-3, GM, CSF
Eo-CFU
EOSINOPHIL GRANULOCYTE
IL-3, GM. CSF. G-CSF
NEUTROPHIL GRANULOCYTE
GM-CFU
G-CFU M-CSF, IL-6, SCF M-
CFU GM, CSF, IL:3, M-CSE
MONOCYTE/MACROPHAGE
LEGEND
BFU: Erythroid Explosive Unit
CFU: Colony Forming Unit
E: Erythrocyte
Eo: Eosinophil
Ep0: Erythropoietin
GEEM: Granule-erythroid-megakaryocyte-macrophage
GM: Granule-monocytic
IL: Interleukin
LIF: Leukocyte inhibitory factor
M: Monocytic
Mast: Mast cell
Mk: Megakaryocyte
SCF: Stem cell factor

Fig. 64.1 General scheme of hematopoiesis.

Fig. 64.2 Compartmental model of hematopoiesis.


Totipotent and pluripotent stem cells (Stem cells)
Committed stem cells
Morphologically identifiable blast cells
Differentiated and functional cells
Proliferation fraction
Self-renewal capacity

Except for lymphocytes, the compartmental model is characterized by:

The progressive loss of the capacity for self-renewal (characteristics of the stem cell compartment).

The extensive capacity for proliferation or amplification within the first three compartments.

The progressive and irreversible specialization from one compartment to the

ERYTHROPOIESIS

Erythropoietic ontogeny begins in the multipotent stem [Colony Forming Granule-Erythroid-Megakaryocyte-Macrophage Unit (CFU-GEMM)]. immediate and common progenitor of the Explosive Unit Erythroid (BFU-E) and the Colony Forming Unit Erythroid (CFU-E), which do not have morphologically identifiable characteristics and are only recognized in vitro, due to their ability to form colonies (see Fig. 64.1).

The identifiable progenitor cells are made up of four stages of maturation, all of them morphologically distinguishable. Each maturation stage represents a new cell division; The first is the proerythroblast, followed by the basophilic erythroblast, the polychromatophilic erythroblast and the orthochromatic erythroblast (Fig. 64.3) .

After these four divisions, the nucleus of the orthochromatic erythroblast becomes pyknotic and unable to synthesize DNA, so they stop dividing and henmatides are formed without a nucleus that is expelled.

The erythrocyte seen in peripheral blood goes through a stage of reticulocytes (without nucleus), which preserve ribosomes and nucleic acids that are stained with supravital dyes and give a filamentous appearance similar to reticulin. These reticulocytes mature in 2-3 days and become erythrocytes or red blood cells (see Fig. 64.3), which live for approximately 120 days, after which they are destroyed by the cells of the reticuloendothelial system, the vast majority in the spleen.

The maturation process requires 5-7 days and erythropoietin (hormone produced mainly by the kidney) plays an important role in erythropoiesis.

The function of the red blood cell is to transport hemoglobin, which is oxygenated at the level of the alveolar-capillary circulation of the lungs and is thus distributed throughout the body; In this way it intervenes in tissue respiration. It also contributes to the transport of carbon dioxide from the tissues to the lungs.

Mature red blood cells are very deformable and at rest are biconcave discs with an average diameter of 8.4 u. Elasticity depends on the volume and surface area ratio, but it is capable of passing through vessels 2 u in diameter. It is equipped with enzymatic systems to use glucose, although it lacks the tricarboxylic acid cycle (Krebs cycle). Its only source of energy is adenosine triphosphate (ATP), obtained by anaerobic glycolysis.

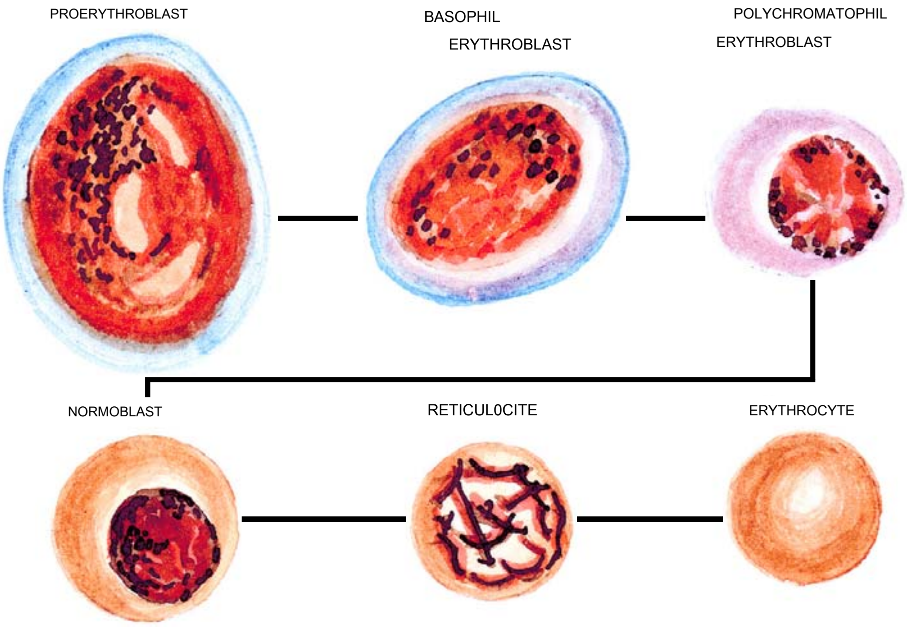


Fig. 64.3 Erythrocytic series (morphologically identifiable cells).

- Embden-Meyerhoff pathway until lactate (90%).

Pentose-phosphate cycle (10%). Abundant nicotinamide adenine dinucleotide phosphate dehydrogenase (NADPH) is formed, which stabilizes glutathione, reducing it.

- Rappaport-Luebering 2,3 diphosphoglycerate cycle.

2,3 diphosphoglyceric acid (2,3 DPG) is formed, which will influence the affinity of hemoglobin for oxygen.

The hemoglobin contained in the erythrocyte comprises approximately 95% of its dry weight or 30% of its wet weight; the rest is water, anions, cations (especially potassium, glucose, etc.). It has a molecular weight of 68,000, is elliptical in shape and is made up of four polypeptide chains (globin), to each of which a heme is attached. Heme belongs to the class of pigments known as porphyrins and is composed of a cyclic tetrapyrrole bonded to a central ferrous ion. Globin polypeptide chains are designated by Greek letters:

alpha (a), beta (B), delta (8), gamma (y), epsilon (ε) and zeta (). Each hemoglobin molecule has four chains, identical in pairs. Alphas and zetas are encoded on chromosome 16; the others, in 11.

- 95-97% is hemoglobin A (HbA) with two alpha and two beta chains (xß).
- HbA2 is less than 3% (two alpha, two delta) (ȍ),.

Other hemoglobins: fetal hemoglobin or HbF (two alpha, two gamma) (y),, Hb Portland (two gamma, two zeta) (YŠ),

LEUKOPOYESIS

Granulocytic series: neutrophils, eosinophils and basophils

It is currently known that although these three series develop in parallel and have common morphological and physiological characteristics, there are well-proven differences in their origin and that to understand them we must refer to the hematopoiesis scheme (see Fig. 64.1).

These series, in their morphologically recognizable expression, are made up of a succession of cells that begin in the myeloblast and end in the granulocyte, a name acquired thanks to the presence of granulations in the cellular cytoplasm and whose staining details vary depending on whether it is an eosinophil, a basophil or a neutrophil: the latter is also called, erroneously, segmented polymorphonuclear, due to the division of the nucleus into a variable number of lobes (figs. 64.4, 64.5 and 64.6).

The immature components of these series are found in the red marrow of the bones, intermixed with the elements of the erythrocytic, megakaryocytic and agranulocytic (lymphocytic, monocytic) series. They mature in that place, transit through the blood and exert their function in the tissues.

The main function of neutrophil granulocytes is phagocytosis, which they carry out due to their richness in proteinases and other enzymes and which is generally facilitated by opsonization (germ or particles covered by an antibody and complement).

Therefore, neutrophils are the fundamental weapon in the fight against microbes. Their number increases significantly in suppurative processes and infectious diseases, situations in which, in addition to the adult neutrophil, younger cells of this cell line (previous maturational stages) appear in the peripheral blood, such as the stab, the juvenile, the myelocyte and the promyelocyte, a hematological detail known as left shift and which are considered elements that appear when bacterial infections are more aggressive.

Basophils contain a group of substances that could be preformed, associated with cytoplasmic granules, or produced during the activation process of said cells, such as: proteinglycans, histamine, proteases, platelet activating factor, metabolites of arachidonic acid and many others, which could have some influence on inflammatory processes.

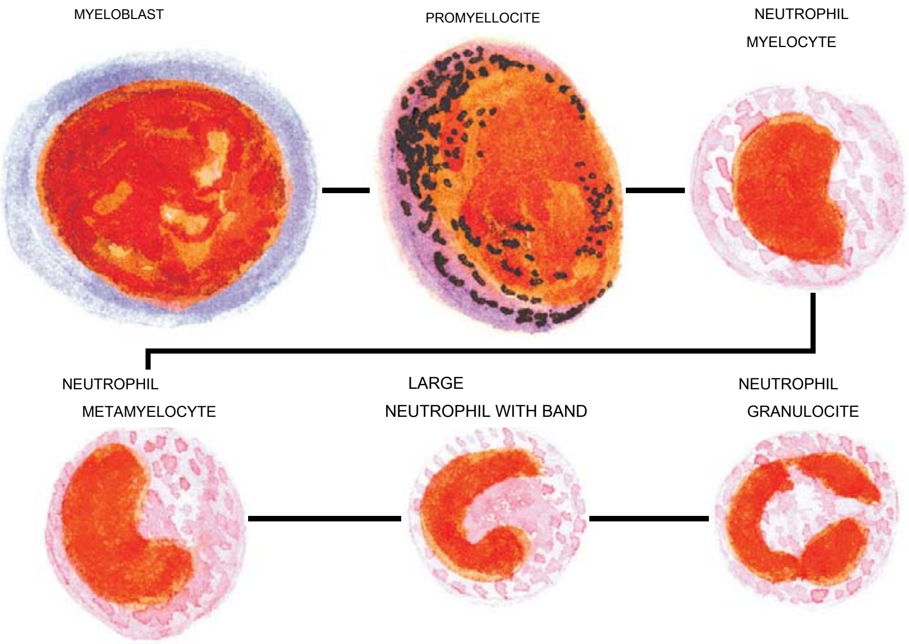
Eosinophils have a certain defense function in the body, especially against some parasites, through the so-called " frustrated phagocytosis", which is nothing more than the discharge of the toxic content of their granules, directly on their objective or target. Furthermore, they may have a beneficial function in canceling some aspects of hypersensitivity reactions through mechanisms such as: the neutralization of histamine and the production of a substance called an eosinophil-derived inhibitor, which prevents the degradation of mast cells.

Agranulocytic series: monocytic and lymphocytic

monocytic series

The bone marrow is the source of neutrophils and monocytes, which circulate through the vascular spaces until their exit to the tissues. Both come from a common parent: the Granule-Monocytic Colony Forming Unit (CFU-GM).

Fig. 64.4 Neutrophil series (morphologically identifiable cells).


MYELOBLAST
PROMYELLOCITE
NEUTROPHIL MYELOCYTE
NEUTROPHIL METAMYELOCYTE
LARGE NEUTROPHIL WITH BAND
NEUTROPHIL GRANULOCITE

The monoblast is the first morphologically identifiable cell, followed by a maturation stage known as the promonocyte and finally, the mature cell:
the monocyte (fig. 64.7), which is the largest of the leukocytes (10-20 u in diameter) and is part of the mononuclear-phagocytic system, exercising the phagocytic function on cellular debris, foreign bodies, plasmodia and mycobacteria.

lymphocytic series

Lymphocytes are mononuclear elements devoid of granules, which are found in the circulating blood, in the lymph, in the bone marrow and in lymphoid tissues, such as lymph nodes and spleen, as well as in subepithelial lymphoid tissues such as tonsils, adenoids, Peyer's patches and appendix.

In mammals and birds there are two different cell populations, the T lymphocyte (thymus-dependent) responsible for cellular immunity and the B lymphocyte (bursa-dependent) responsible for the production of antibodies or immunoglobulins, which is the so-called humoral immunity (fig. 64.8).

The B lymphocyte (large or medium) takes its name because, in birds, it is produced in the bursa of Fabricius,

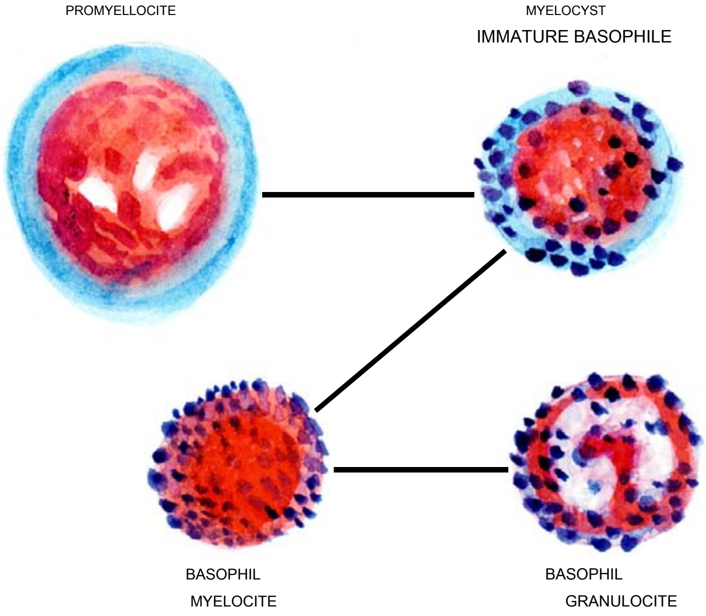

PROMYELLOCITE
MYELOCYST
IMMATURE BASOPHILE
BASOPHIL MYELOCITE
BASOPHIL GRANULOCITE

Fig. 64.5 Basophilic series (morphologically identifiable cells).

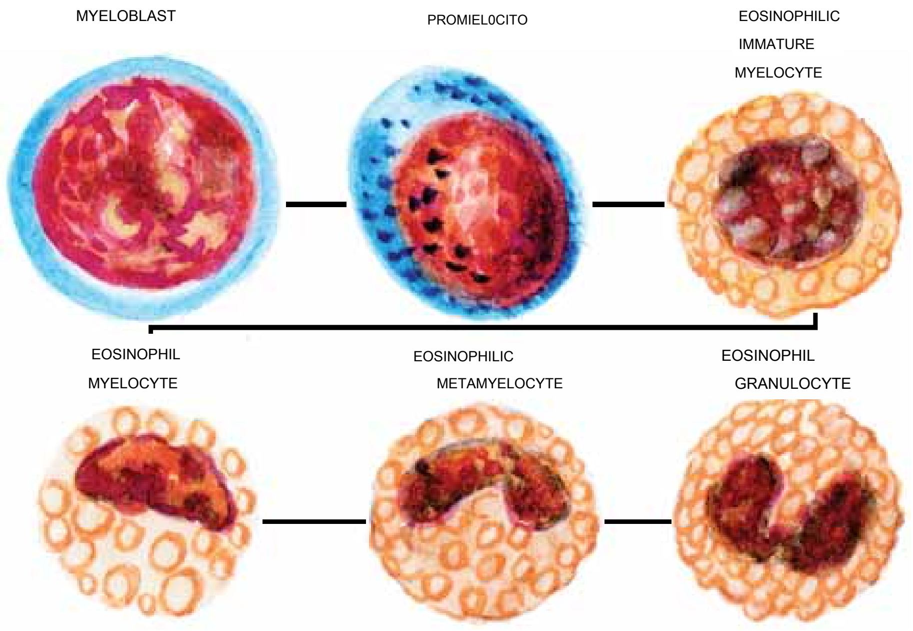

MYELOBLAST
PROMIEL0CITO
EOSINOPHILIC
IMMATURE
MYELOCYTE
EOSINOPHIL
MYELOCYTE
EOSINOPHILIC
METAMYELOCYTE
EOSINOPHIL
GRANULOCYTE

Fig. 64.6 Eosinophilic series (morphologically identifiable cells).

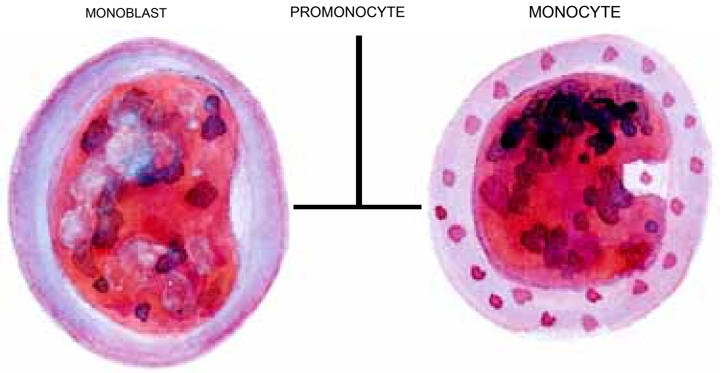

MONOBLAST
PROMONOCYTE
MONOCYTE

Fig. 64.7 Monocytic series (morphologically identifiable cells).

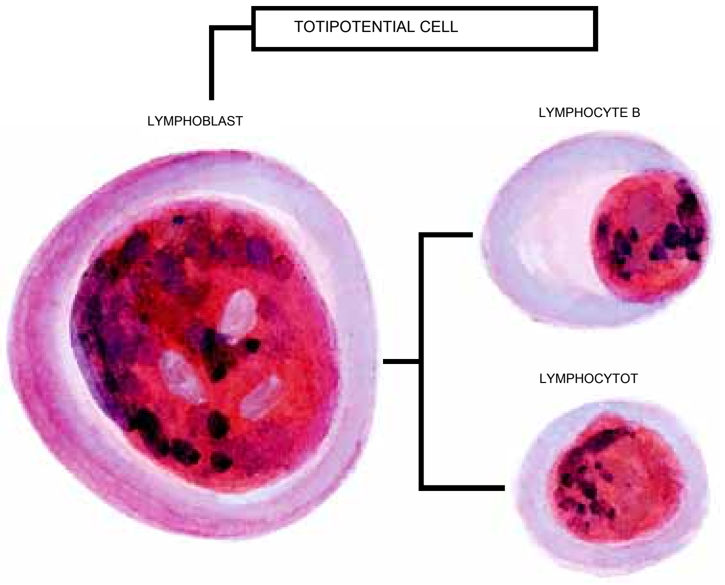

TOTIPOTENTIAL CELL
LYMPHOBLAST
LYMPHOCYTE B
LYMPHOCYTOT

Fig. 64. 8 Lymphocytic series.

lymphoid formation that does not exist in man. However, this type of lymphocyte is formed in the bone marrow and represents almost its entire lymphoid population, which is why the name B lymphocyte can be used.

The lymphocyte in its origins must go through a whole series of stages that begin in the totipotent cells, continue with a committed precursor (precursor cell of the lymphocytic lineage) and from there it differentiates into two cellular elements: pre-T and pre-B, from which the morphologically identifiable stages lead to the T and B lymphocyte. This entire differentiation process requires the intervention of multiple cytokines.

MEGAKARIOPOYESIS

Promegakaryoblasts come from the Megakaryocyte Colony Forming Unit (CFU-Mk) demonstrable in in vitro cultures; These cells are transitional or intermediate and are not recognized by their morphology in bone marrow samples, if specific markers are not used (examples: von Willebrand factor (VWF), glycoproteins IIb and IIa, platelet peroxidase). Morphologically recognizable megakaryocytes exist in four well-defined stages of maturation, which differ by their morphology, antigenic expression, enzyme content and DNA content. From the cytoplasmic fractionation of megakaryocytes, platelets arise, which are the smallest elements of the blood (2-3 u in diameter) and which are necessary for the maintenance of normal hemostasis and have an important role in the processes pathologies of thrombosis and in metastatic cancer (Fig. 64. 9).

PLASMA CELLS

The normal plasma cell has a spherical or ellipsoidal shape, it is a large cell with abundant bluish protoplasm and a round or oval nucleus, located eccentrically, that contains large masses of chromatin arranged like the spokes of a wheel.

Its origin is well known; They are derived from the B lymphocyte, as shown in the hemopoiesis diagram.

Normal plasma cells make antibodies, which are nothing more than immunoglobulins. Currently, there are five types of immunoglobulins that are designated with the acronym Ig, adding the letters G, A, D, M and E, depending on the type.

Immunoglobulins make up the most complex system of proteins that we know today in the field of biochemistry. They have common and particular characteristics. A common characteristic is the composition, since they are formed by light and heavy polypeptide chains, linked by disulfide bridges. It is precisely the heavy chains that are responsible for the nomenclature for immunoglobulins, described above.

In pathological situations, plasma cells can proliferate abnormally, giving rise to so-called plasma cell dyscrasias (for example, multiple myeloma), diseases that cause a certain monoclonal gammopathy (a paraprotein, or functionally altered immunoglobulin, is secreted, despite having a chemical structure similar to the normal immunoglobulin). In these cases some details could be found

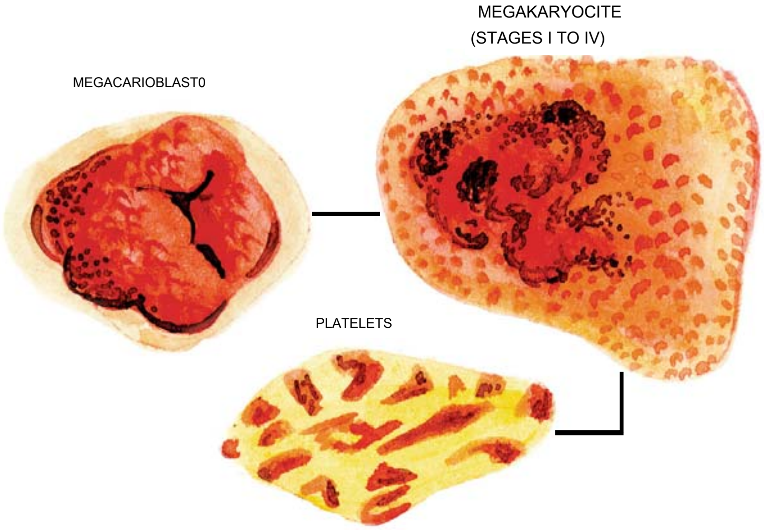


Fig. 64. 9 Megakaryocytic series.

atypical cellular morphology: presence of mitosis, intracytoplasmic vacuoles, plasmacytes with nucleoli (plasmoblasts) and

MAIN SYMPTOMS AND SIGNS

The most frequent symptoms and signs of the hemolymphopoietic system are

- Cutaneomucosal pallor.
- Glossitis.
- Bone pain.

Purple.

- Bruises.

Increase in volume of the spleen and lymph nodes.

- Neurological manifestations.

CUTANEOMUCOUS PALENESS

This sign was previously treated in skin disorders (volume 1, Chapter 25). It is the most important sign of anemia.

GLOSSITIS

It is seen in pernicious anemia. The tongue is swollen, irritated, the lingual papillae atrophy, and the patient complains of burning and painful discomfort.

The jasmine red color that the tongue acquires can extend to the entire back of it or be limited to the edges. Sometimes it is accompanied by stomatitis.

BONE PAINS

Sometimes bone sensitivity increases, especially in the sternum, with the patient experiencing pain when the bones are percussed. It is observed in multiple myeloma, leukemias and bone metastases, fundamentally.

PURPLE

Purpura or purpuric lesions are multiple spontaneous hemorrhages of the skin and mucous membranes that do not disappear under vitro pressure (fig. 64.10).

They are mainly observed in platelet or vascular alterations, and were already described along with the rest of the skin alterations, in volume 1, Chapter 25.

HEMATOMAS

They are larger blood collections than ecchymoses and are observed in trauma, in a deficiency of coagulation factors, or in excess of anticoagulants.

The hematoma is not a purpura.

INCREASE IN VOLUME OF THE SPLEEN AND GANGLIA

Both splenomegaly and lymphadenopathy are signs of relevant importance in hematology and imply an important invasion of the lymphoreticular tissue. When we study the physical examination of the lymphatic system and spleen (Volume 1, Section I, Chapter 17), we describe their semiotechnics; and when studying the splenomegalic and adenic syndromes (in the next chapter) we will insist on their semiotechnics, diagnosis and pathophysiology.

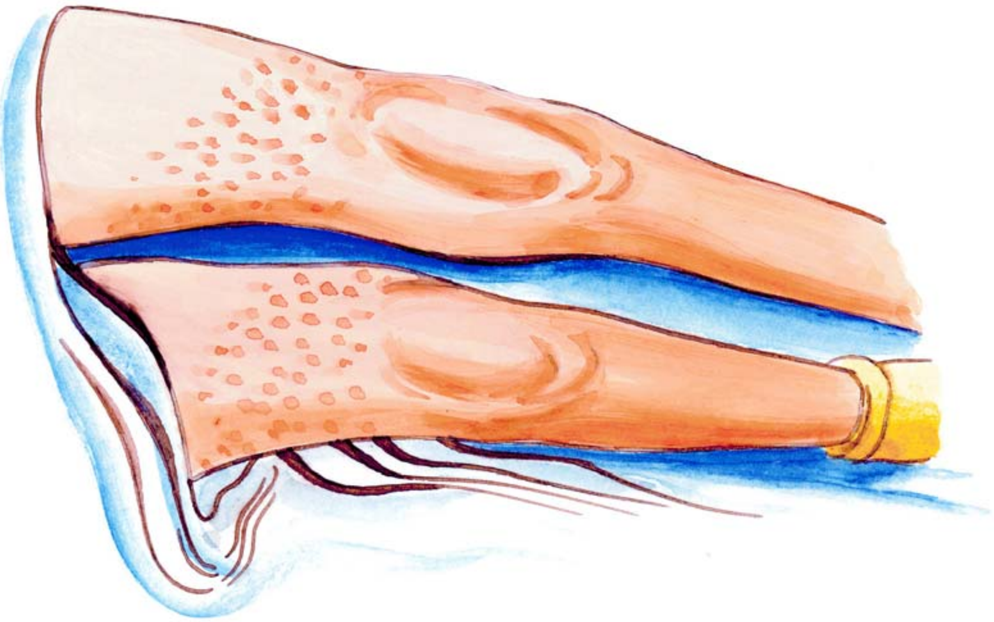


Fig. 64. 10 Purple.

NEUROLOGICAL MANIFESTATIONS

In anemias due to DNA synthesis disorders due to vitamin B deficiency, there are degenerative processes of the lateral and posterior cords of the spinal cord (combined sclerosis) and peripheral nerves, which are manifested by subjective symptoms and physical signs. Among the former, paresthetic sensations (tingling, numbness of the limbs), weakness, muscle rigidity and even psychological disorders may appear.

Among the physical signs we sometimes find: decreased sensitivity (hypoesthesia), when pricking certain areas of the body with a pin, decreased vibratory sensitivity to the tuning fork and ataxia, which implies an injury to the spinal white matter through which the fibers that conduct superficial and deep sensibilities, and the motor fibers, pass.

A bone tumor such as multiple myeloma can cause spinal cord compression.

The existence of thrombotic or hemorrhagic manifestations in the nervous system can give rise to the most varied syndromes, according to the extent and topography of the process.

MAIN SİNDROMES OF THE HEMOLYMPHOPOIETIC

65

Introduction

We will limit ourselves, given the characteristics of this text, to briefly studying those syndromes that occur most frequently in the clinic: these are:

- Anemic syndrome.
- Adenic syndrome.
- Splenomegalic syndrome.
- Purpuricohemorrhagic syndromes.

ANEMIC SYNDROME

CONCEPT

The set of symptoms and signs determined by anemia is called anemic syndrome.

Anemia appears when the number of red blood cells, hemoglobin or both decreases to levels below the physiological limit. In men, the normal number of red blood cells is considered to be 4,500,000 to 5,000,000/mm² and hemoglobin between 13 and 15 g, with the normal hematocrit being 45%. In women, the physiological values are: red blood cells, between 4,000,000 and 4, 500,000/mm'; hemoglobin, between 12 and 14 g% and hematocrit 41%.

NORMAL PHYSIOLOGY OF ERYTHROPOIESIS

It is necessary to remember the normal physiology of erythropoiesis to better understand the factors involved in the production of erythrocytes and the pathophysiology of the anemic syndrome.

The multipotent stem cell CFU-GEMM gives rise to erythrocytes following a series of steps, which we already studied in the previous chapter.

Erythropoietin produced in the kidney initiates the passage to the proerythroblast. The following mechanisms are carried out with the help of coenzymes (B, and folic acid), iron and vitamin B,.

Vitamin B, comes from food, binds in the mouth to an R protein, subsequently binds to intrinsic factor to finally absorbed in the terminal ileum and then transported by transcobalamin II (TC II).

Folic acid is absorbed in the jejunum and also comes from vegetables and fruits.

Iron has an endogenous reuse and only small doses from the outside are necessary; It is absorbed at the duodenum and transported by transferin. In the synthesis of hemoglobin chains, amino acids such as glutamic acid, lysine, etc. participate.

PHYSIOPATHOLOGICAL CLASSIFICATION

There are many classifications proposed for the study of anemias, but we will follow the one that takes into account the pathophysiological alterations of the erythrocyte progeny, which seems most rational to us.

In this classification, seven types of anemia are studied:

1. Disorders in the multipotential and unipotential stem cell.
2. Disorders in DNA synthesis.
3. Disorders in heme synthesis.
4. Disorders in globin synthesis.
5. Disorders in the enzymatic synthesis of red blood cells.
6. Disorders due to external interference in the functions of the bone marrow.
7. Disorders due to interference in the survival of red blood cells.

Disorders in the multipotential and unipotential stem cell

These disorders can affect the multipotential stem cell and in that case all the series that derive from it are affected (erythrocytic, granulocytic and megakaryocytic), manifesting in the clinic by the appearance, in addition to anemia, of leukopenia and thrombocytopenia, as occurs in aplastic anemia; In these cases, one series is sometimes affected more than the other and in the medullogram a depression will be found with a decrease or absence of the youngest cells.

Another example of this type of clinical picture is the frequency of infectious processes of different types when the granulopoietic series is affected and the presence of bleeding disorders (purpura, gingivorrhagia, etc.) when the involvement of the megakaryopoietic series predominates.

The disorder can be unipotential, such as when there is a deficiency of erythropoietin, in these cases the red or erythrocytic series is affected, and only anemia appears; An example of this type of anemia is observed in chronic renal failure.

Deficiencies in the thyroid and other hormones, as well as physical or chemical injuries (radiation, lead, benzenes) also cause this type of anemia.

Disorders in DNA synthesis

For its synthesis, DNA needs vitamin B, and folic acid, which act as coenzymes. As we already said, vitamin B is absorbed in the terminal ileum in the presence of intrinsic factor and folic acid in the jejunum.

When there is a deficiency of these factors, an alteration occurs in nuclear maturation that is expressed by the appearance in the peripheral blood and marrow of a large cell called a megaloblast and, on some occasions, the megakaryopoietic series may also be affected with a reduction in platelets, which is accompanied by purpuric manifestations.

These clinical pictures are observed in patients with nutritional deficiency, non-absorption of vitamin B, gastric or ileal resections, antibodies against intrinsic factor, parasitic pathology that affects the small intestine and the use of certain drugs.

As typical examples of this group, we point out pernicious anemia and malabsorption syndromes.

Disorders in heme synthesis

Hemoglobin contains four heme groups with an iron atom located in the center of a porphyrin structure, a ferroprotoporphyrin structure that gives hemoglobin the red color typical of a red blood cell.

Anemias due to disorders in heme synthesis usually occur due to disorders in iron absorption, in cases of gastric resections, celiac disease, gastrojejunostomy; due to consumption during lactation, pregnancy, neoplasia and due to iron loss (most frequently) during chronic bleeding from ulcers, hiatal hernia, colon carcinoma, during parasitism due to hookworm, necator, etc., and chronic gynecological bleeding.

An important characteristic of these anemias is the presence of small erythrocytes, with little hemoglobin (hypochromic microcytic anemias).

Disorders in globin synthesis

Globin is part of the hemoglobin molecule and is made up of two pairs of polypeptide chains and its synthesis is regulated by different genes, as expressed in the previous chapter.

In this group, qualitative defects in the α or β chains and quantitative differences are studied, which result in anemias due to hemoglobinopathies. In our environment, the most common is sickleemia or sickle cellemia, secondary to a synthesis disorder.

of hemoglobin, where glutamic acid is replaced by valine and this is accompanied by the formation of textoid bodies and the deformity of the red blood cells, which take the shape of a banana or half-moon (sickling cell), which contributes to their easy destruction when exposed to hypoxic or anoxic situations.

Disorders in the enzymatic synthesis of red blood cells

In this type of anemia, the disorder occurs when certain enzymes that are involved in the synthesis of red blood cells are deficient, which produces a decrease in globular resistance and destruction of red blood cells. These anemias are more common in childhood and were formerly known as congenital hemolytic anemias.

Other times the present mechanism is related to a congenital disorder as is usually seen in microspherocytosis, where the erythrocytes adopt a spheroidal shape.

We also have non-spherocytic and enzymatic congenital hemolytic anemias, such as, for example, anemias due to glucose 6-phosphate dehydrogenase and pyruvatokinase deficiency.

Disorders due to external interference in bone marrow functions

The cause of this type of anemia is secondary to the invasion, replacement or displacement of marrow cells by other types of external tissues, as occurs in the case of distant metastases or neoplasms and in marrow infiltration by leukemic cells or myeloma cells. These types of anemia have been designated as myelophthisic anemias.

Disorders due to interference with the survival of red blood cells

It may happen that all the steps mentioned above for the maturation of erythrocytes develop normally, but that they are lost when they enter the peripheral blood, due to hemolysis due to the action of antibodies, due to splenic sequestration or due to blood loss, as observed in acute hemorrhages. Transfusion reactions mediated by isoantibodies should also be included here.

In all these cases there is hyperplasia in the bone marrow that tries to compensate for the losses. The erythrocyte is normal, but its survival is shortened.

In summary, from the pathophysiological point of view, the clinical manifestations common to all anemias depend on the hypoxia that it implies and the body's ability to adapt to the new condition. situation. The underlying disease or cause of the anemia will enrich the clinical picture with the symptoms and signs dependent on it.

Chronic anemias are better tolerated than acute ones (sudden onset), so the intensity of the symptoms does not always reflect the degree of anemia;
We have an example in sickle cell patients, who sometimes have very low hemoglobin levels with few symptoms.

Pernicious and nutritional anemia can also occur with very low numbers of red blood cells and hemoglobin, and the patient can continue walking and performing normal tasks that do not require physical effort.

SYNDROMOGRAPHY OR POSITIVE DIAGNOSIS

In the diagnosis of anemia, both clinical data and those provided by laboratory tests must be considered.

A clinical history collected with the smallest details is very useful to suspect, not only anemia, but its type, which can only be specified with the help of laboratory investigations. Sometimes the diagnosis is obvious, such as when anemia appears after an acute hemorrhage, but it is often very difficult to determine its cause.

Clinical picture

The following subjective symptoms and physical signs must be considered:

a) Cutaneomucosal pallor. It is the most relevant sign and is observed above all in the conjunctival and oral mucosa, in the earlobe, in the palm of the hand and in the nail bed. It can take on different shades: yellowish pallor of hemolytic anemia; alabastrin, for acute hemorrhages;
straw yellow, malignant neoplasms and pernicious anemia. Sometimes trophic disorders of the skin and mucous membranes are observed.

b) Asthenia.

c) Tachycardia and decreased pulse width, which can lead to peripheral vascular collapse in cases of acute hemorrhage. In cases of chronic anemia, functional murmurs, palpitations and precordial tightness can be detected.

d) Dyspnea due to anemic anoxia.

e) Headache, vertigo, lipothymia, instability, restlessness and drowsiness.

f) Ringing in the ears, amblyopia, vision of floaters, etc.

g) Sensitivity disorders produced by degeneration of the posterior and lateral cords of the spinal cord, as in pernicious anemia.

Depending on the cause of the anemia, the clinical picture may be accompanied by splenomegaly, hepatomegaly, lymphadenopathy, hemorrhagic manifestations or other signs.

us.

Complementary exams

Count and characteristics of red blood cells.

Hemoglobin.

- Hematocrit.

Corpuscular constants.

-Reticulocyte count.

- Leukogram.

Platelet count.

- Medulogram.
- Other specials.

ADENIC SYNDROME

CONCEPT

The increase in volume of the lymph nodes produces a group of symptoms and signs, which constitute the adenic syndrome. Lymph node disease is called adenopathy. This syndrome is characterized by enlarged lymph nodes (adenomegaly), which can be localized or generalized.

SYNDROMEGENESIS OR PHYSIOPATHOLOGY

Lymphocytes and reticuloendothelial cells are the most important cellular elements of the lymph nodes.

While the former (Ty B lymphocytes) intervene in immune phenomena, the reticuloendothelial cells (histiocytes and macrophages) have as their main function the phagocytosis of any substance foreign to the body; For example, germs coming from an area whose lymphatics are tributary to those nodes.

The nodes therefore carry out a mechanical filtration of the lymph, eliminating foreign particles, bacteria and cellular remains that may have reached it from the various areas drained by the lymphatic vessels.

The increase in volume of the lymph nodes (adenomegaly) can be produced by an increase in the number and size of the lymph follicles due to the proliferation of their cellular elements or by infiltration of cells foreign to the node. This infiltration can It can be due to metastasis of a carcinoma, leukemia, or produced by polymorphonuclear cells, as observed in inflammatory processes (adenitis).

In hoarding diseases (thesaurismosis) macrophages are loaded with lipid or other substances.

SYNDROMOGRAPHY 0 POSITIVE DIAGNOSIS

The examination of the lymph nodes was explained in volume 1, Chapters 4 and 17. It is suggested that they be reviewed before addressing the positive diagnosis of adenic syndrome. As expressed in these chapters, in normal adults the lymph nodes are not palpable or only a slight increase in volume is detected (banal adenomegaly). To verify the existence of enlarged lymph nodes, it is necessary to previously know the location of the lymph node chains. Remember that these are found in the lateral regions of the neck, in the armpits, epitrochlears and inguinals (see Fig. 17.1).

Secondly, it is necessary to specify the senmiographic characteristics of this syndrome:

-Location.

- Temperature.
- Sensitivity.

If there is necrosis.

Size.

- Symmetry.
- Mobility.

Consistency.

- If it adheres to the deep planes.

Other aspects that we must take into account are the following:

- Lymphadenopathy can be localized or generalized.

The increase in lymph nodes located in the deep regions (abdominal, mediastinal) is discovered by the compression it produces on neighboring structures. For example, lymphadenopathy that occurs in the mediastinum can produce a superior cava syndrome, those of the hepatic hilum, an ictero-ascitic syndrome, and lumboaortic lymphadenopathy, an inferior cava syndrome, with edema in both lower limbs.

When it is accompanied by heat, there is redness of the skin and there is pain, it suggests an inflammation of the lymph node; It can give rise to periadenitis, become necrotic and produce fistulas, as can be seen in tuberculosis. In chronic infections there is usually no edema or pain.

- The lymph nodes of chronic leukemias and lymphomas can be large, symmetrical, firm and mobile, but not very sensitive.

When it is a metastatic carcinoma, they are stony, non-painful and non-movable.

The different lymph node chains of the neck were already listed in volume 1, Chapter 4 and illustrated in figure 4.22, of that chapter. For educational purposes and for the reader's convenience, the different lymphatic areas of the cervical region are shown again (fig. 65.1). The different steps to follow to perform palpation are also described and illustrated (fig. 65.2).

In step 1, the nodes in the submental region can be explored. This is followed by the exploration of the submandibular region (step 2). The preauricular and parotid areas are then palpated (step 3). In step 4, the high jugular lymphadenopathy is located, then the spinal lymphadenopathy (step 5) to continue with the low jugular lymphadenopathy and finish in the supraclavicular regions (step 6-7-8).

Figure 65.3 illustrates how to examine the axillary lymph nodes. It shows the position in which the patient's upper limb is maintained and the hand of the explorer who palpates, on the wall of the armpit, to apply pressure with the palms, from top to bottom, which, in the case of lymphadenomas, allows evaluating its semiographic characteristics. The patient's upper limb should not be raised too much, to avoid exercise.

There is a lot of tension in the armpit.

DIFFERENTIAL DIAGNOSIS

Not all neck tumors are always an expression of enlarged lymph nodes and other causes must be differentiated, such as the presence of lipomas, branchial cysts, aneurysms, and others.

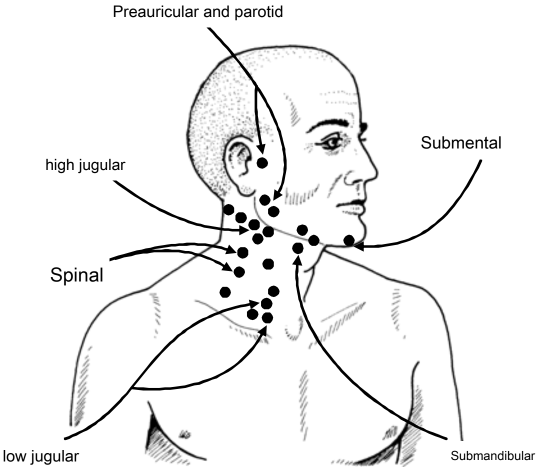


Fig. 65.1 Lymphatic areas of the cervical region.

ETIOLOGY

When faced with a patient with lymphadenopathy, several considerations are important.

The first is age, the second is knowing that benign processes that cause lymphadenopathy decrease with age.

Reactive hyperplasia of lymphoid tissue in response to infection or inflammatory processes is characteristic of children.

Lymphadenopathy of the supraclavicular regions is associated with intrathoracic or intra-abdominal malignant processes.

The progressive enlargement of the nodes for more than several weeks, associated with fever, chills, night sweats and weight loss suggests a systemic disease, such as tuberculosis, a mycosis or a malignant lymphoproliferative disease.

Among the causes of lymphadenopathy and depending on its location, we have the following groups:

1. Cervical lymphadenopathy:
   - Infections of the face.
     Dental abscesses.
     External otitis.
   - Bacterial pharyngitis.
   - Infectious mononucleosis.
   - Cytomegalovirus infection.
   - Adenovirus.
   - Rubella.
   - Toxoplasmosis.
   - Malignant hemopathies.
   - Lymphomas.
   - Carcinoma of the nasopharynx or larynx.
2. Supraclavicular lymphadenopathy:
   - Intrathoracic and intra-abdominal malignant diseases.
   - Breast cancer.
     Supraclavicular lymph node or sentinel lymph node Virchow on the left side, as an expression of a abdominal gastric neoplasia.
3. Axillary lymphadenopathy:
   - Cat scratch fever.
   - Metastasis of breast carcinoma.
   -Lymphangitis.
   - Suppurative lesions of upper limbs.
4. Inguinal lymphadenopathy:
   Repeated trauma to lower limbs.
   Minor infections of the genitals and lower extremities (cellulitis).

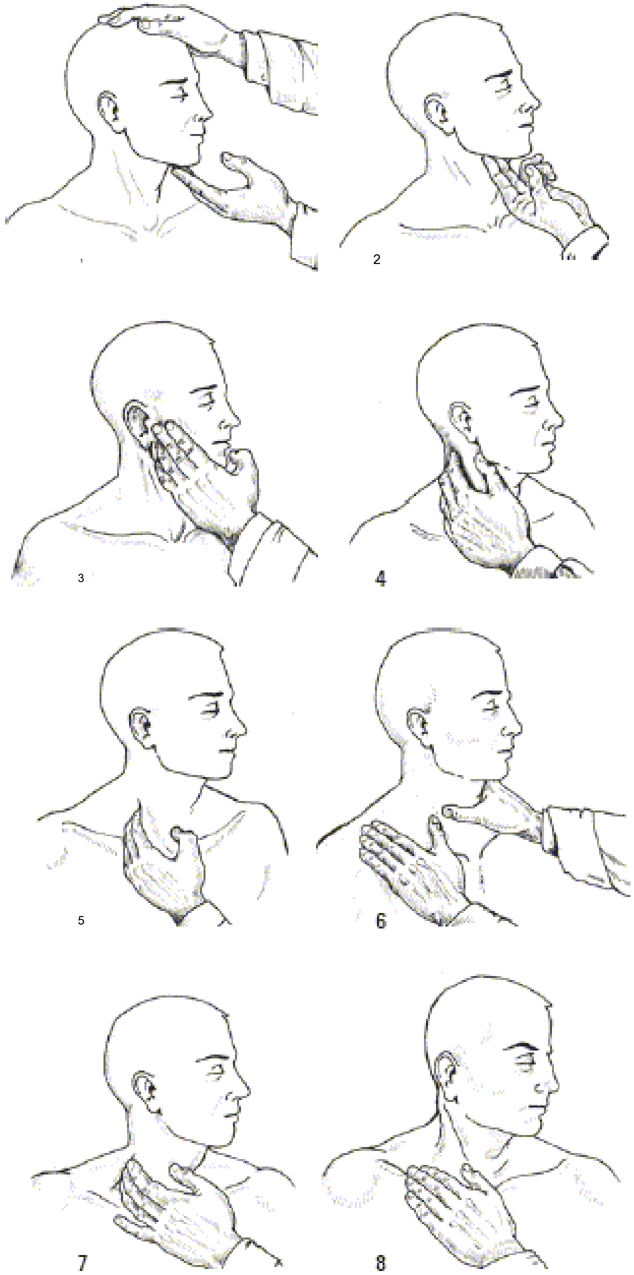


Fig. 65.2 Technique for palpation of the different lymphatic areas of the cervical region.

Infections of the perineum.

Venereal infections (syphilis, chancre, genital herpes, lymphogranuloma venereum).

- Squamous cell carcinoma of the penis or vulva.

5. Generalized lymphadenopathy:

Systemic diseases: systemic lupus, erythematous rheumatoid arthritis.

- Drug ingestion: phenytoin, allopurinol. hydralazine,

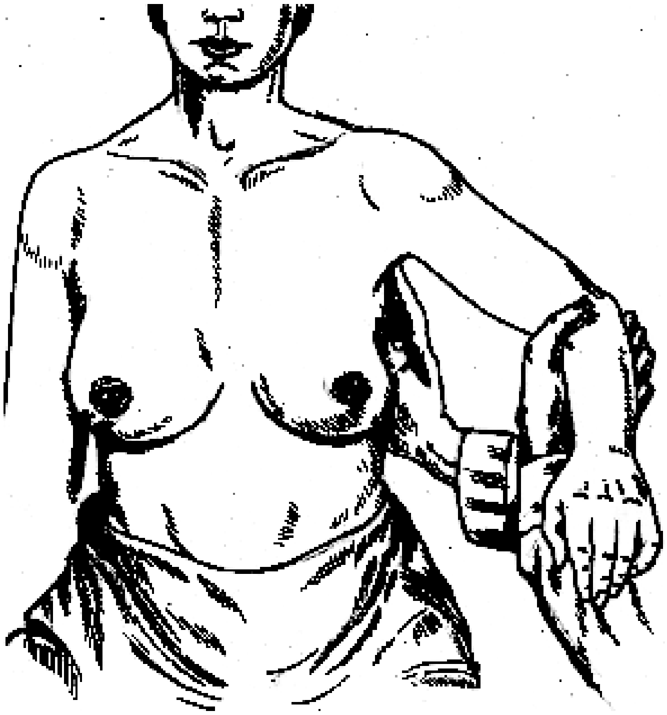


Fig. 65.3 Technique for palpation of axillary lymph nodes.

Infections: mononucleosis, Epstein-Barr virus, cytomegalovirus, toxoplasmosis, AIDS, histoplasmosis.

SPLENOMEGALIC SYNDROME

CONCEPT

This is the name given to the set of symptoms and signs that appear with an enlarged spleen.

The spleen can be considered a large lymph node and, therefore, has the same functions as the nodes and increases in volume in the same processes that we have considered in lymphadenopathy. In the spleen, the function of sequestering damaged or aged formed elements of the blood is of great importance. It also serves as a platelet reservoir. In fetal life it is a hematopoietic organ, a function that it loses after birth, unless in pathological conditions of extramedullary hematopoiesis, in which case its volume increases greatly.

As an organ that is part of the portal circulation, its size can also increase in cases of hypertension of the portal circuit.

SYNDROM0GRAPHY 0 POSITIVE DIAGNOSIS

Clinical picture

Data obtained by anamnesis

The subjective symptoms in diseases of the spleen are very inconstant. Most of the time they are reduced to a simple sensation of tension or weight, if the spleen is greatly enlarged. If there is inflammation of the peritoneum (periesplenitis) the pain can be intense, persistent, fixed and not radiating. In splenic embolism (septic valvular heart disease, for example) the pain is very acute, collapsing.

The pain in splenic conditions is dull and is located in the splenic region; It can be the precursor symptom of splenomegaly that sets in more or less quickly. A more acute pain, which worsens with breathing, indicates the presence of inflammation of the capsule (periesplenitis). This pain, when very intense, usually radiates to the left supraclavicular region, through the phrenic, and to the shoulder and back on the same side, through the third and fourth cervical roots.

Collaterally, we must bear in mind that pain in the splenic region, with or without the aforementioned irradiations, accompanied by shock or not, with or without contracture of the parietal muscles of the upper abdominal quadrant, and with the characteristic semiology of an internal hemorrhage, will always suggest a rupture of the spleen. Spontaneous rupture originates in a diseased spleen (chronic malaria, leukemia, typhoid fever, septic endocarditis). A healthy spleen ruptures as a result of abdominal trauma, which can occur days after the trauma occurs. Therefore, a history of trauma or the existence of one of the splenic conditions mentioned is of great diagnostic help.

Large splenomegaly, as in chronic malaria, leukemia or myeloid metaplasias, for example, cause discomfort and sometimes even pain. Huge splenomegaly causes diffuse abdominal discomfort and pain due to compression of the stomach and intestines, as well as functional disorders of these organs (anorexia, mnetheorism, nausea, vomiting, diarrhea or constipation, among other symptoms).

Data from physical examination of the spleen

The physical examination procedures to detect splenomegaly (inspection, palpation and percussion) were described and illustrated in volume 1, Section I, Chapter 17 and their prior review is recommended. Some other useful elements will be detailed here. Auscultation of the spleen, although it must be performed Always, to detect friction in cases of perisplenitis, in general it provides little data, which is why it was not mentioned in volume 1 and is not mentioned in this chapter.

Data obtained by inspection

When the spleen hypertrophies it grows downward, because the diaphragm contains it above. Furthermore, the increase in visceral weight overcomes the weak means of support available to it, so that the organ descends towards the abdominal cavity.

To become visible, it must increase considerably in size, as is only observed in chronic leukemias, chronic malaria, agnogenic myeloid metaplasia or Banti syndrome, mainly.

These large splenomegaly cause a bulge of the belly, which crosses the abdomen obliquely from the left hypochondrium to the right iliac fossa.

Data obtained by palpation

Palpation is the great method of exploration of this organ. A palpable spleen is abnormal. When the spleen is palpable, it must be descended or enlarged. Splenic ptosis is recognized by its normal size and by the possibility of carrying it below the costal margin, even during inspiration, which cannot be done with an enlarged spleen. In the absence of ptosis, the spleen becomes palpable only when it doubles or triples in size.

The spleen is recognized by its edge, with perfectly palpable notches (notches).

Some spleens are very soft (those of patients with typhoid fever, for example) and require very careful palpation to be appreciated.

Remember the characteristics that the spleen presents, for its palpatory recognition:

1. Location below the costal margin between the IX and XI ribs.
2. Superficial situation immediately below the abdominal wall.
3. Anterior edge with incisions or notches.
4. Respiratory mobility wide and diagonally towards the navel.

Palpation of the spleen allows us to explore its location, shape, size, sensitivity, consistency, surface, mobility and, occasionally, beating and rubbing.

The situation has already been discussed previously.

The shape of the spleen is preserved in most splenomegaly. Abscesses, cysts and tumors often deform it.

As for the size, when the magnification is small it is only verified by percussion. Acute splenomegaly, as occurs in typhoid fever and other sepsis, usually causes moderate increases.

In these cases the spleen is palpable only on inspiration or slightly extends beyond the costal margin.

Chronic splenomegaly gives greater increases.

The spleen does not have much sensitivity. It hurts in perisplenitis processes caused by infarctions, abscesses, subcapsular hematomas, malignant tumors and cysts. In acute splenomegaly, distension of the capsule can cause dull, mild pain in the splenic region, which the patient sometimes reports spontaneously.

The consistency of the spleen depends on the nature of the splenomegaly, its size and its duration. Typically, the older and larger the splenomegaly, the greater the consistency. Acute splenomegaly is soft. The spleen of amyloid degeneration may have a woody consistency.

Abscesses and cysts of the spleen may have a soft or even fluctuating consistency. But since they are rare tumors, one should always doubt whether what is palpated is the spleen.

The surface of most splenomegaly is uniformly smooth. " Bumps" (prominences) can appear in heart attacks, abscesses, and cystic and malignant tumors.

The spleen has mobility due to the action of breathing, gravity and the action that can be manually imprinted on it. Its means of fixation and its contact with the diaphragm make it move widely with this muscle. This type of mobility is one of the most important characteristics to recognize the spleen, and it is lost when it acquires extraordinary dimensions or when the diaphragm is paralyzed due to abdominal, thoracic or muscle causes.

Occasionally, a heartbeat may be felt when palpating the spleen. A ptotic or large spleen may beat when resting on a very pulsating aorta. By placing the patient face down or in a position on all fours, the heartbeat disappears when that contact is lost. Splenic pulsation has been reported in aortic insufficiency.

Also occasionally, rubs may be palpated on the splenic surface. They are signs of perisplenitis, abscesses and infarctions,

The provocation of cough (splenic cough) when palpating the spleen has been reported. It is a simple visceromotor reflex with no diagnostic value.

Data obtained by percussion

Although the techniques for percussion of the spleen have already been described in Section I, volume 1, and in this chapter we have already recalled its characteristics for palpatory recognition, it is worth insisting, for the adequate obtaining of pathological data by percussion, that said organ is located between the IX and XI ribs, along the of the spine. The left lung is interposed in its upper third and therefore eludes percussion. Splenic dullness corresponds to the anteroinferior two-thirds of the organ, usually directly attached to the thoracic wall (fig. 65.4). Also keep in mind that if the dullness of the spleen measures more than 7 cm in height, in the direction of the body axis, in the average-sized adult, it should already be considered pathological.

DIFFERENTIAL DIAGNOSIS

It should be done with tumors of the left hypochondrium:

1, Kidney tumors, abscesses or perirenal inflammations.

2. Adrenal tumors.
3. Carcinomas of the splenic flexure of the colon or fecalomas therein.
4. Pancreatic cysts or carcinomas.
5. Carcinomas or sarcomas of the stomach.

The location of the tumor, its characteristics, as well as the accompanying symptoms corresponding to the affected organ, are useful for differential diagnosis. We will not delve into it, since this description corresponds to Internal Medicine.

CLINICAL INTERPRETATION OF SPLENOMEGALY

Once the spleen has been explored and all its location conditions, size, shape, sensitivity, consistency, mobility, etc., are known, and the differential diagnosis has been made, it is necessary to perform semiological interpretation to reach the etiological diagnosis of splenomegaly.

The clinician usually makes a first differentiation: whether it is a recently enlarged spleen (acute spleen) or a long-standing process (chronic spleen). This problem is solved with:

a) Data provided by the general clinical picture.

b) Data on the physical conditions of the spleen itself.

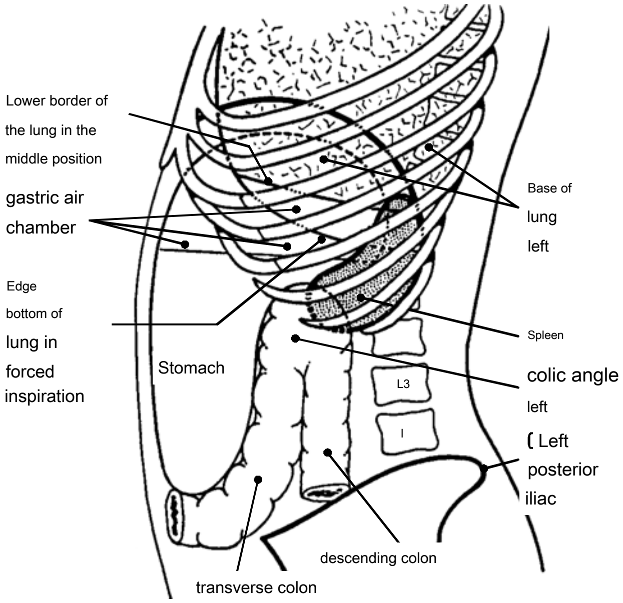


Fig. 65.4 Position of the spleen of a normal individual, in the right lateral decubitus position.

The former will be provided by the meticulous study of the history and by the complete and thorough examination of the patient, including laboratory investigations.

Regarding the physical conditions of the spleen, it will be determined if it is an acute case, in which the spleen, generally, is of a more or less soft consistency and its enlargement is not very pronounced; or if it is a chronic case in which the spleen, almost always, is hard in consistency and its size varies, depending on the processes; or if they are very large spleens that widely exceed the costal margin and even overflow the umbilical scar.

PATHOPHYSIOLOGY AND ETIOLOGY

Splenomegaly can be one more manifestation of a process of florid symptomatology by other systems, or constitute in itself the prevalent symptom, to which others are added, closely related from the physiopathogenic point of view.

Thus we will have:

1. Splenomegaly in the course of infections:
    a) Acute: systemic infections, such as infectious diseases, typhoid fever, septicemia, infectious mononucleosis and others.
    b) Subacute: bacterial endocarditis, malaria, parasitic infections such as trypanosomiasis, schistosomiasis, etc.
    c) Chronic: tuberculosis, congenital syphilis.
2. Congestive splenomegaly: portal hypertension, thrombophlebitis of the splenic vein and others.
3. Tumor splenomegaly: lymphomas and cysts.
4. Splenomegaly due to hoarding: Niemann-Pick Gauchery disease, etc.
5. Hyperplastic splenomegalies: leukemias and hemolytic anemias and others.

Let us remember that the spleen is a hematopoietic organ and that it increases in volume in myeloid metaplasia.

Splenomegaly can lead to hypersplenism, which is further characterized by:

1. Anemia, leukopenia and thrombocytopenia, isolated or in combination.
2. Marrow with normal or increased cellularity.
3. Correction of the blood condition by splenectomy.

Splenectomy relieves only one aspect of the basic disorder and rarely cures the underlying disease that gave rise to the splenomegaly.

There are several theories to explain hypersplenism, such as: increased sequestration of formed elements in the blood, bone marrow inhibition, and production of antibodies by the spleen, but their explanation is beyond the limits of this book.

PURPURICHEMORRHAGIC OMES SYNDR

CONCEPT

This is the name given to those processes in which blood escapes from the vascular system, without apparent cause or there is a disproportion between the cause and the intensity of the bleeding. Hemorrhages can occur in the skin and mucous membranes (as in the case of purpura), in the subcutaneous cellular tissue, in the internal cavities of the body (pleura, abdomen), in the joints or in traumatized points.

We will study the so-called hemorrhagic diatheses or blood dyscrasias that occur when the hemostasis mechanism is disturbed.

GENERAL PHYSIOLOGY OF HEMOSTASIA

Before studying the pathophysiology and diagnosis of hemorrhagic syndromes, we will consider the mechanism of hemostasis (Fig. 65.5).

Hemostasis is the stopping of bleeding, in which platelets, vessels and blood clotting intervene. The action of platelets and vessels has also been called primary hemostasis.

The incorporation of coagulation results in secondary hemostasis.

To prevent blood leakage from its vascular bed, the arteriolocapillary wall must have normal resistance and contractility, and platelets must be normal in

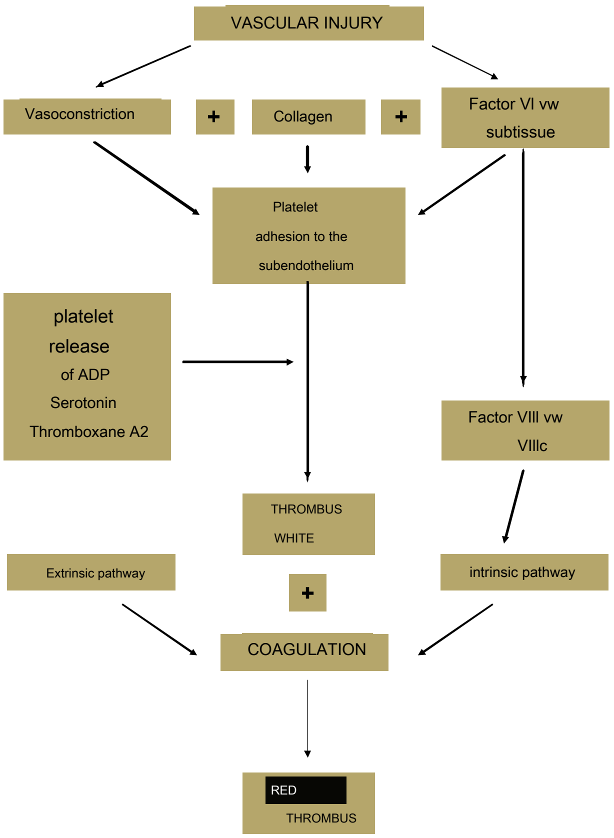


Fig. 65.5 General diagram of hemostasis

quality; That is, a useful clot or thrombus must be produced quickly, all of which is reinforced by the blood coagulation process, to form the tail of the thrombus or red thrombus. To control this process, the body has well-defined mechanisms, such as the action of anticoagulants, the fibrinolytic system and antiplatelet agents.

When a vessel is damaged for any reason, hemostasis begins through the vasoconstriction already mentioned, which is followed by the adhesion of the platelets to the exposed subendothelium, with the presence of collagen tissue, as well as factor VIIIvw, which is produced in the subendothelium, where one part remains and the other is incorporated into the blood circulation and joins the coagulant factor VIII, to form the complex factor VIIvw-VIIc.

The adhesion of the platelet is followed by the release of different substances such as ADP, serotonin and thromboxane A, (a powerful platelet aggregator), which lead to the formation of the white thrombus, which occludes the damaged vessel. To control this stage, prostacyclins with vasodilatory action will be produced at the level of the endothelium, stimulation of platelet adenyl cyclase and the transformation of ATP into cAMP that prevents ADP from stimulating platelet aggregation.

Finally, the platelet aggregation process is followed by the coagulation process, which has an extrinsic stimulation pathway and an intrinsic one, which give rise to the production of thrombin, with the formation of fibrin monomers, which encompasses the figured elements of the blood, and the activation of factor XIII (XIIa), which contributes to forming a firm clot, the last stage of hemostasis.

The factors involved in blood clotting are the following:

- Factor I (fibrinogen).
Factor II (prothrombin).
Factor III (thromboplastin).
Factor IV (calcium).
- Factor V (labile factor).
Factor VII (stable factor).
Factor VIIIc (clotting factor VIIII).
- Factor IX (PTCo plasma thromboplastin component).
X factor (Stuart-Prower factor).
Factor XI (PTA or plasma thromboplastin history).
Factor XII (Hageman factor).
- P.K.: Prekallikrein.
- KIN: Kininogen.

NORMAL MECHANISM OF COAGULATION

For the red clot or thrombus to form, the presence of fibrin is necessary, which originates from fibrinogen through the action of thrombin. The latter is formed from its precursor, prothrombin, through the interaction of activated factors X and V (Xa and Va), calcium and the platelet surface (Fig. 65. 6).

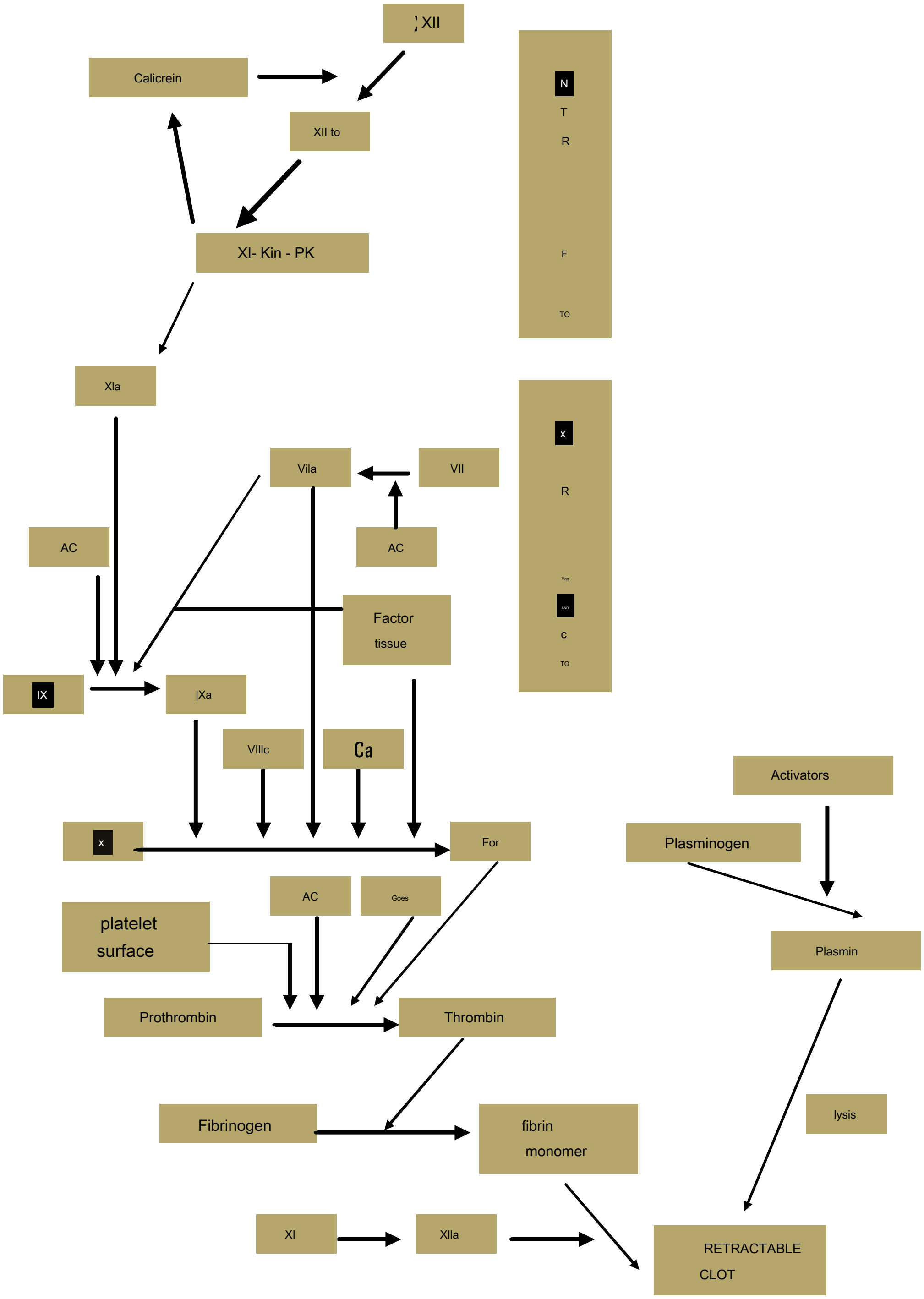
Factor X is activated (Xa) by the action of factor IXa, which is activated in the intrinsic system, by the action of factor

According to some authors, the activation of this complex is only accepted as an in vitro reaction, measurable in the laboratory using the partial thromboplastin time (PTT).

Factor IX is activated in the extrinsic pathway, through factor VIIa. Thrombin generated extrinsically can induce the binding of factor VII complex to platelets concomitantly and activate factor VIIIc to participate in the intrinsic pathway.

Once the vessel is repaired, the fibrin formed is lysed by the action of plasmin (fibrinolysin).

Plasminogen is activated to plasmin by the action of activators.


¦ XII
Calicrein
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Activators
Plasminogen
AC
Goes
platelet
surface
Plasmin
Prothrombin
Thrombin
lysis
Fibrinogen
fibrin
monomer
XI
XIIa
RETRACTABLE
CLOT

Fig. 65.6 Normal coagulation mechanism.

PHYSIOPATHOLOGICAL CLASSIFICATION

- Hemorrhagic diatheses due to disorders in the plasma coagulation mechanisms and excess circulating anticoagulants.
- Hemorrhagic diathesis due to platelet alterations.
- Hemorrhagic diathesis due to vascular alterations.
- Hemorrhagic diathesis of complex etiology.

Bleeding diathesis due to disorders in plasma coagulation mechanisms AND due to excess circulating anticoagulants

This group includes pathological entities whose mechanism is a delay in the formation of the red fibrin clot and bleeding that appears shortly after primary hemostasis (platelets and vessels) occurs.

Grouped here are: deficiencies of plasma factors, studied in the normal coagulation mechanism; bleeding that occurs due to excess circulating anticoagulants; those due to increased fibrinolysis, secondary to different causes, and intravascular coagulation, due to consumption of factors.

Hemorrhagic diathesis due to platelet disorders

Included in this group are dyscrasias produced by a quantitative or qualitative deficiency of platelets, due to a defective release of different components.

Also included is the deficiency of platelet membrane glycoproteins (Ib-IX), which produces a disorder of platelet adhesiveness.

Hemorrhagic diathesis due to vascular alterations

They occur as a consequence of a weakness in the vessel wall or a defect in platelet adhesiveness at the level of the endothelium, due to a deficiency or abnormal formation of factor VIIIvw.

Hemorrhagic diathesis of complex etiology

Those dyscrasias are grouped in which the mechanism of bleeding production is complex, since several factors intervene.

SYNDROMOGRAPHY 0 POSITIVE DIAGNOSIS

The identification of any bleeding disorder requires a careful anamnesis that allows investigating family history, the mode of onset (spontaneous or post-traumatic) of the bleeding phenomenon, the association with an infectious disorder or another known disease, the history of the administration of any anticoagulant or the ingestion of a medication that could be considered toxic, etc.

When faced with bleeding, we must answer these three questions:

1. Is the bleeding caused by a local condition, a hemorrhagic diathesis, or a combination of the two factors?
2. If it is a consequence of a diathesis, which of the factors, plasma, platelet or vascular, is responsible?
3. What is the etiology?

To answer the first question, it is important to do a thorough examination of the bleeding area to rule out a local condition; In addition, you must use the help of specialists (otolaryngologists, gynecologists, urologists, gastroenterologists, etc.). When making a positive diagnosis, you must be sure that the bleeding is caused by a diathesis.

In cases such as hemophilia, if the notion of hereditary data is not known, and if the history of previous bleeding is missing from the anamnesis, its symptoms can be confused with those of other diseases. Thus, a deep hematoma can be confused with an abscess; hemorrhage from a large joint (knee) can lead one to mistakenly think that it is joint tuberculosis, deforming arthritis, etc.; If there is phantom hematuria, a kidney tumor can be considered; hematemesis and melena may raise suspicion of a peptic ulcer, etc.

Sometimes, patients come to the clinic due to excessive bleeding from small wounds and hematuria, melena, bruises and petechiae. Then, the clinician has to recognize the existence of a generalized hemorrhagic state.

A tooth extraction or a nasopharyngeal operation that causes bleeding for more than 48 hours, or that requires a blood transfusion, is an unequivocal sign that the patient has a blood dyscrasia.

Finally, it should be remembered that bleeding caused by an obvious local condition can be precipitated by an unsuspected bleeding disorder, and in turn bleeding in a patient with a bleeding diathesis can be precipitated by a local condition.

Regarding the answer to the second question, that is, the pathophysiological diagnosis of the hemorrhage, this can be suspected according to the type of bleeding.

Purpuric syndrome is formed by the existence of spontaneous hemorrhages of the skin and mucous membranes (petechiae, ecchymoses). and in these cases one should not think of plasma coagulation defects, but of platelet or vascular conditions.

In thrombopathies, we frequently find a normal coagulation time, with a positive loop test, prolonged bleeding time, poorly retractile clot, and low platelet count; in thrombocytopenias, clotting time may be prolonged.

In platelet disorders, petechiae are the most striking sign, and ecchymoses generally do not exceed 2 cm in diameter. Mucous membrane bleeding is common. Bleeding from wounds begins immediately, persists for less than 48 hours, and

They rarely recur.

In vascular disorders, bleeding is limited to the skin, usually in the form of petechiae and ecchymoses, and the characteristics of bleeding from wounds are the same as those of platelet disorders.

Abnormal hemorrhages due to plasma diathesis that cause a coagulation defect (unlike the bleeding of hemorrhagic purpura) are not limited to the skin and mucous membranes and are generally related to trauma or tissue damage. When these damages affect the body surface, obvious hemorrhages occur; If there is no solution to continuity, repeated bleeding can produce a hematoma, sometimes enormous, for example, in the joint cavity.

Bleeding from wounds tends to begin after a period of several hours, persist for more than 48 hours, and recur after apparent hemostasis has occurred. This is not absolute, since in purpura we sometimes find the existence of deep bleeding, and in dyscrasias due to a coagulation defect, there are very rare cases of purpuric syndrome; But in general, bleeding on the surface guides the study of platelets and blood cells.

glasses.

We find the answer to the third question by evaluating the anamnesis, the physical examination and certain laboratory tests.

In thrombocytopenic purpura, the study of the medullogram is important, since idiopathic thrombocytopenic purpura is megakaryocytic and secondary cases often present with amegakaryocytic medullogram.

Acquired vascular conditions that are accompanied by other types of elementary skin lesions (erythema, exanthema, edema, etc.) are frequently due to immunoallergic disorders (Schonlein-Henoch disease).

A long history of bleeding that began in childhood is an important manifestation that the disorder is congenital in nature.

Because some blood dyscrasias are hereditary, it is necessary to do a family history. careful, investigating even the grandparents. In cases of rare types of coagulation defect and in hemophilic families, in which there are women who bleed, consanguinity should be considered.

All coagulation defects observed in plasma diatheses can produce the same clinical picture, so this is not capable, on its own, of determining the possible etiology of the condition.

Even hemarthrosis is not pathognomonic of hemophilia. We must insist, then, on the genetic study.

If the patient is male and the rest of the males in the family are affected, but not the females, we can think of hemophilia A, due to factor VIIIc deficiency, or Christmas disease (PTC). If bleeding exists in women, it must be another type of diathesis, since the previous two are the only recessive ones linked to sex.

Congenital afibrinogenemia is generally the result of a marriage between cousins, and appears to be inherited recessively; that is, autosomal recessive, although cases that are inherited as autosomal dominant have been reported.

Deficiencies in factors VIIc and IX can be considered as sex-linked recessive, and deficiencies in the other factors as autosomal recessive.

In congenital factor XIII deficiencies, in addition to the hemorrhagic symptoms, there is a delay in wound healing.

If there is no family history and the bleeding has occurred late, this condition does not appear to be hereditary and an immediate cause must be sought, for example, an excess of anticoagulants, which gives rise to a hemophiloid state.

Other congenital diatheses that cause hemophiloid symptoms are deficiencies in fibrinogen, prothrombin, factors V and VII, Rendu-Osler disease, von Willebrand disease and Ehlers-Danlos syndrome.

Fibrinogen deficiency may also appear in severe liver disease, active fibrinolysis or in cases of disseminated intravascular coagulation.

Prothrombin deficiency is seen in liver disease, obstructive jaundice, malabsorption syndromes and in all cases in which vitamin K deficiency occurs.

Factor V deficiency (parahemophilia) can also occur in liver disease and intravascular coagulation processes.

The deficiency of factor VII is seen, above all, during treatment with oxycoumarins and also in liver diseases.

Hemorrhagic telangiectasia, von Willebrand disease and Ehlers-Danlos syndrome are familial and also cause deep bleeding. In

The first, there may be epistaxis, hemoptysis, etc., but in these cases telangiectasias are observed on the lips, tongue, nose, skin or around the nails of the fingers of the hand, which pale with pressure, which does not happen with petechiae.

In von Willebrand disease there is a deficiency of factor VIlvw, as well as alterations in platelet adhesiveness and aggregation, and bleeding time is prolonged.

Deep bleeding and wound bleeding may occur in Ehlers-Danlos syndrome, but the hyperelasticity of the skin and hyperflexibility of the joints help in the diagnosis.

It is important to note that in the majority of alterations of coagulation factors there is no true deficiency of them, but rather a structural alteration of their molecules that causes them to lose their biological procoagulant properties.

The importance of a good clinical history must be highlighted, since the diagnosis of many blood dyscrasias is fundamentally clinical, and, furthermore, the selection of the laboratory tests required for an exact diagnosis depends on a correct clinical assessment.

ETIOLOGY

1. Plasma hemorrhagic diathesis:

   a) Fibrinogen and factor XII deficiency.

   b) Deficiency of the factors that alter prothrombin time: II, V, VII and X.

   c) Hemophilias. Hemophilia A, when there is a deficiency of factor VIII; hemophilia B, if the decreased factor is factor IX and hemophilia C, when the low concentration is factor XI. In von Willebrand disease, a qualitative platelet disorder is associated with a decrease in factor VII.

   d) Diathesis due to excess circulating anticoagulants.

2. Platelet hemorrhagic diathesis:

   a) Quantitative platelet alterations.

   Thrombocytopenias (decreased number of platelets). Thrombocytopenias can be primary, as in idiopathic thrombocytopenic purpura (ITP), which have an immunological origin, or secondary due to physical, chemical, infectious or systemic causes, among others.

   Thrombocythemia (increase in the number of platelets, but with alterations in their quality). The permanent increase in platelets is called thrombocythemia; It has been considered a myeloproliferative syndrome, and as such it can transform into another of the serious diseases of the syndrome, for example, certain types of leukemia.

   Thrombocytosis. It is the transient and reactional increase in platelets, such as that which occurs after a splenectomy.

   b) Qualitative platelet alterations (thrombocytopathies). In qualitative platelet disorders there may be a normal number of platelets, but there are alterations in adhesion or aggregation, or there is a deficiency of platelet factor number 3.

3. Vascular hemorrhagic diathesis:

   a) Congenital. Rendu-Osler disease or hemorrhagic telangiectasia, Ehlers-Danlos syndrome, and others.

   b) Acquired.

   Anaphylactoid purpura (Schonlein-Henoch disease).

   Infectious.

   Avitaminosis (scurvy).

   Chemical agents.

   Systemic vascular disorders. Example: polyarteritis nodosa.

   Metabolic disorders. Example: uremia.

   Senile purple.

   Pigmentary purples.

   Fictitious purples.

   Others.

URINARY SYSTEM. ANATOMY, PHYSIOLOGY, SEMIOLOGY AND COMPLEMENTARY EXAMS

66

ANATOMY AND PHYSIOLOGY

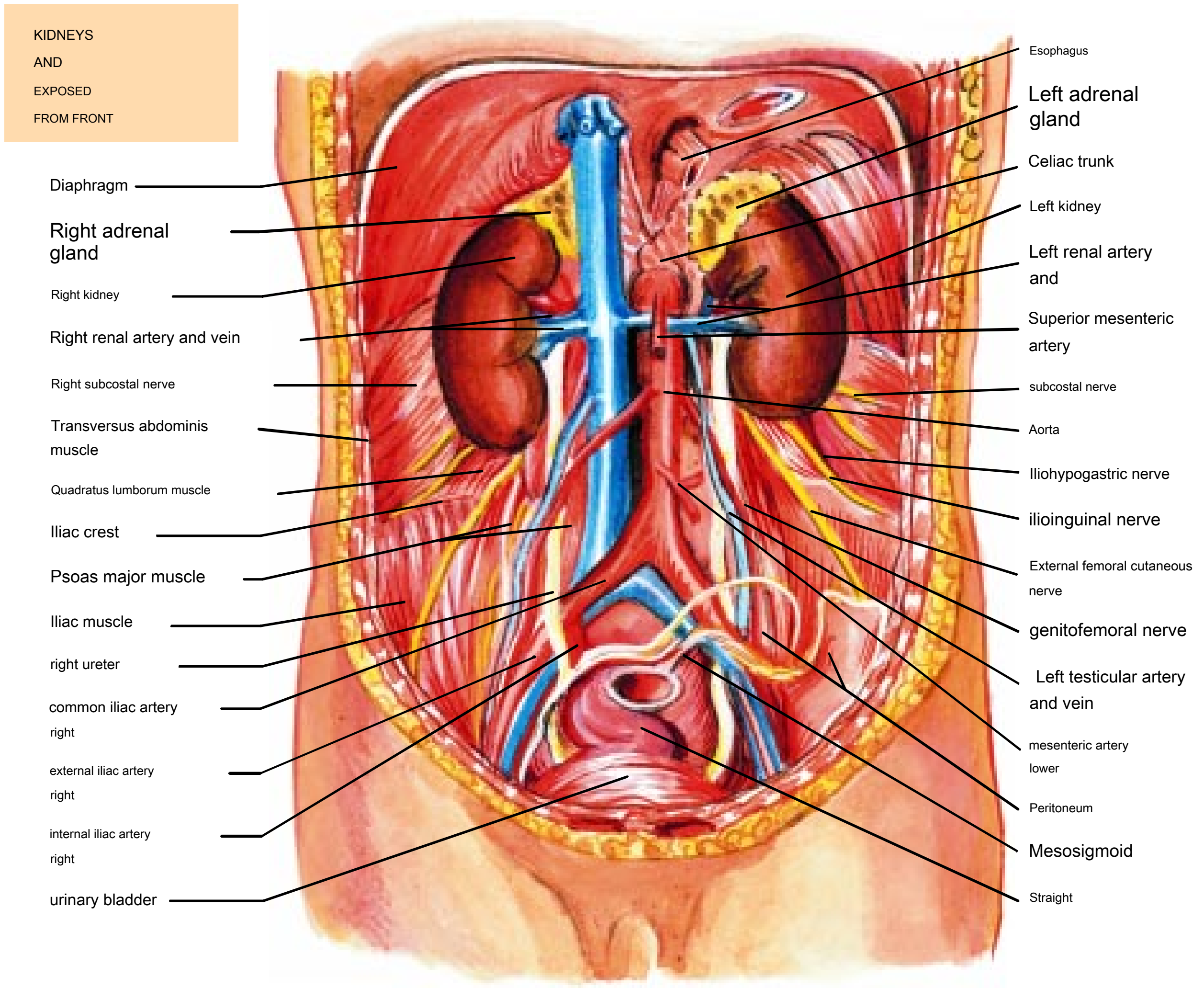
The kidneys are retroperitoneal organs located on both sides of the spinal column (D11 to L3); the right one is slightly descended, which is attributed to the presence of the liver. Normally they should not be palpated. They are covered by a fibrous capsule that is firmly adhered and at the same time easy to separate. They are bean-shaped, with the notch or hilum facing inward. On average, each measures 12 x 6 x 3 cm and weighs 150 g (fig. 66.1).

The excretory system extends from the renal parenchyma to the bladder and from this to the outside. It consists of the calyces (major and minor), the renal pelvis, the ureter, the bladder and the urethra.

The morphological and functional unit of the kidney is the nephron (Fig. 66.2). It is estimated that each kidney is provided with approximately one million nephrons. The nephron, whose average length is about 5 cm, is made up of a vascular component, the glomerulus, a cluster of interconnected capillaries, interposed between two arterioles: the afferent and the efferent, which approach it through its external or vascular pole, and a tubular component, formed by the following elements: Bowman's capsule, which together with the glomerulus, to which it surrounds and attaches its visceral leaf, constitutes the corpuscle. Malpighian, located in the renal cortex; the proximal convoluted tubule, originating in the internal or urinary pole of the glomerulus, also located cortically, the loop of Henle, with a descending branch that enters to a variable depth into the renal medulla and an ascending branch that goes towards the vascular pole of the glomerulus to continue, insensibly, with the distal convoluted tubule, flexuous and also located in the cortex, which ends in the collecting duct, which descends through the renal medulla and when it joins with others it originates the Bellini ducts, which flow into the excretory system, in the minor calyx, at the level of the papilla.

The papilla constitutes the truncated vertex of each Malpighian pyramid, which are medullary formations delimited laterally by the extensions made by the cortex in depth (Berlin columns). The bark cap that covers the base of the Malpighian pyramid is known as the Ferrein pyramid and forms a renal lobe with it.

Each kidney has between 6 and 18 lobes.


KIDNEYS
AND
EXPOSED
FROM FRONT
Diaphragm
Right adrenal gland
Right kidney
Right renal artery and vein
Right subcostal nerve
Transversus abdominis muscle
Quadratus lumborum muscle
Iliac crest
Psoas major muscle
Iliac muscle
right ureter
common iliac artery
right
external iliac artery
right
internal iliac artery
right
urinary bladder
Esophagus
Left adrenal gland
Celiac trunk
Left kidney
Left renal artery
and
Superior mesenteric
artery
subcostal nerve
Aorta
Iliohypogastric nerve
ilioinguinal nerve
External femoral cutaneous
nerve
genitofemoral nerve
Left testicular artery
and vein
mesenteric artery
lower
Peritoneum
Mesosigmoid
Straight

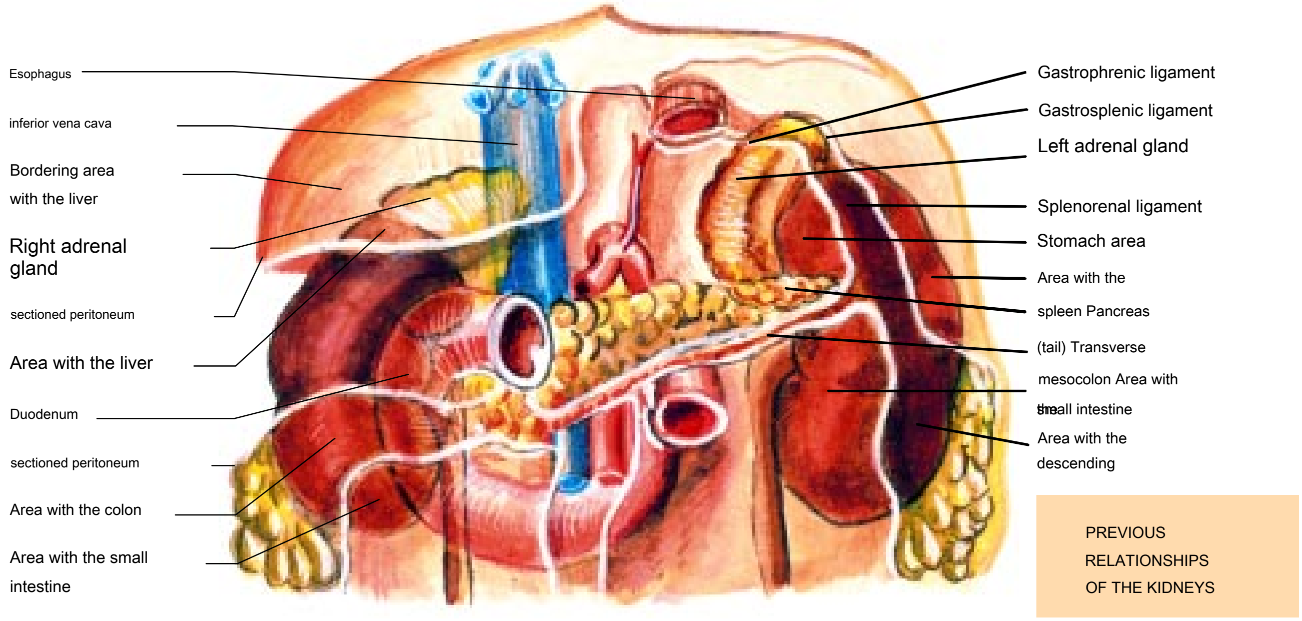

Esophagus
inferior vena cava
Bordering area
with the liver
Right adrenal gland
sectioned peritoneum
Area with the liver
Duodenum
sectioned peritoneum
Area with the colon
Area with the small
intestine
Gastrophrenic ligament
Gastrosplenic ligament
Left adrenal gland
Splenorenal ligament
Stomach area
Area with the
spleen Pancreas
(tail) Transverse
mesocolon Area with
the
small intestine
Area with the
descending
PREVIOUS
RELATIONSHIPS
OF THE KIDNEYS

Fig. 66.1 Anatomical relationships of the kidney.

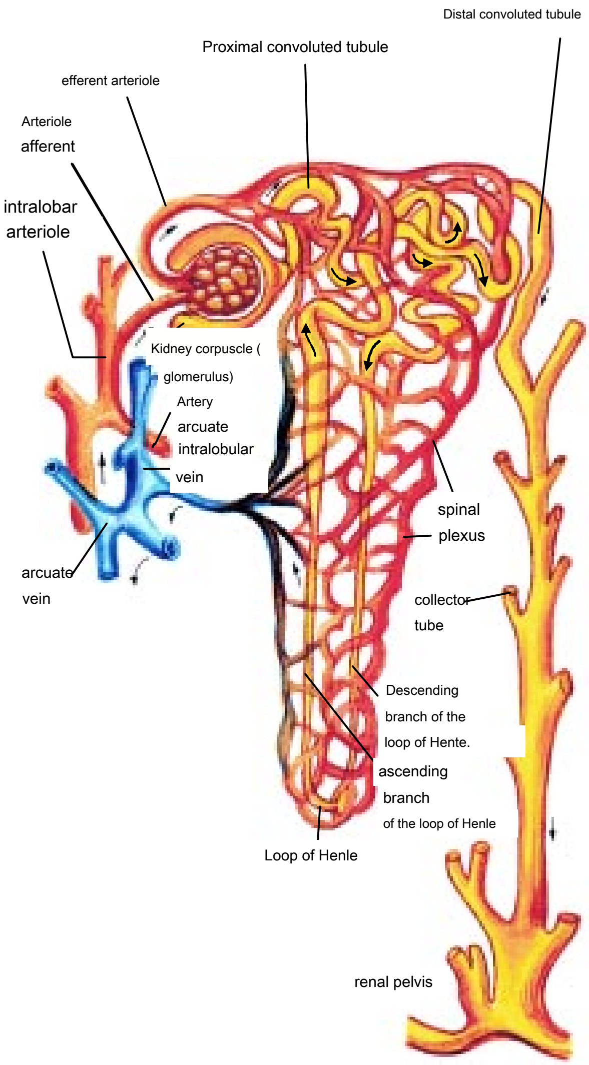

Distal convoluted tubule
Proximal convoluted tubule
efferent arteriole
Arteriole
afferent
intralobar
arteriole
Kidney corpuscle (
glomerulus)
Artery
arcuate
intralobular
vein
arcuate
vein
spinal
plexus
collector
tube
Descending
branch of the
loop of Hente.
ascending
branch
of the loop of Henle
Loop of Henle
renal pelvis

Fig. 66.2 Nephron: anatomofunctional unit of the kidney.

The function of the glomerulus is to form a protein-free filtrate from the plasma. Every minute, about a quarter of the cardiac output (renal fraction of cardiac output) reaches the kidneys, which constitutes the so-called renal blood flow. For an average adult, a cardiac output is estimated to be around 5 L/min and a renal blood flow of 1,200 mL/min. Its plasma fraction (55% of renal plasma flow) is called renal plasma flow and represents about 650 mL/min. One fifth of the renal plasma flow is filtered at the level of the glomerulus and gives rise to glomerular filtrate or ultrafiltrate (Fig. 66.3). This is approximately 125 mL/min, 7.5 Lh and 180 L/24 h. This ultrafiltered Plasma is produced in the glomerulus without the expenditure of energy, through an entirely physical process whose formula is the following:

P= PP,+P)

where:

P: filtration pressure;
P,: capillary hydrostatic pressure;
P: oncotic pressure of plasma proteins;
P; capsular (intratubular) pressure.

Using the normal values of these pressures we will have:

P. = 70 – (30 + 20) =20 mm

Although important tension changes occur, constant filtration is maintained within certain limits by virtue of adjustments in the tone of the afferent and efferent arterioles (renal autoregulation).

The basic function of the proximal convoluted tubule is the active reabsorption (with energy expenditure) of 85% of the glomerular filtrate, which is actively transported from the tubular lumen to the peritubular capillaries.

The function of the loop of Henle is to create, through the so-called countercurrent mechanism, a hypertonic medullary interstice, favoring the facultative reabsorption of water at the level of the collecting ducts.

In the vascular pole of the glomerulus, the distal convoluted tubule and the afferent arteriole are found in close relationship, with notable modifications of the cells of the vascular wall (macula dense) and the arteriolar wall (juxtaglomerular cells), to constitute a group called the juxtaglomerular apparatus (Fig. 66.4). Its main function is the production of renin in response to a drop in blood volume, blood pressure or sodium depletion. Renin is an enzyme whose action triggers the renin-angiotensin-aldosterone mechanism, of great importance in the regulation of blood pressure and hydromineral and acid-base balance. Indeed, angiotensin is a powerful vasoconstrictor and aldosterone promotes sodium retention and elimination of potassium and hydrogen ions, at the level of the distal convoluted tubule. The juxtaglomerular apparatus also participates in the production of erythropoietin, in response to hypoxic stimuli.

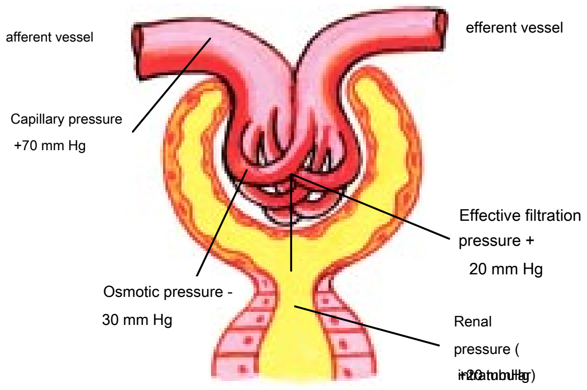


Fig. 66.3 Pressure gradients that determine glomerular filtration.

The reabsorption of water in the collecting duct is controlled by antidiuretic hormone,* of hypothalamic origin (supraoptic and paraventricular nuclei), which regulates the permeability of the basement membrane. The osmotic difference in favor of the interstitium (hypertonic) explains the reabsorption of water, the antidiuretic effect and the emission of concentrated, hypertonic urine.

In summary, the kidneys, through their mechanisms of glomerular filtration and tubular reabsorption and excretion, play a central role in maintaining homeostasis, representing, according to the happy expression of a famous researcher (Homer Smith), the main basis of our physiological freedom. In fact, kidney function goes beyond the narrow framework of urine formation, since it participates in the regulation of the volume of body fluids, electrolyte concentration, acid-base balance, blood pressure and erythropoiesis, in addition to guaranteeing the excretion of the final products of protein catabolism. If we consider that only around 1-1.3 L of urine is eliminated daily, compared to the formation of 180 L of glomerular filtration, we must admit with Oken that the job of the kidney is not to form urine but to prevent it from being formed.

SEMIOLOGY OF THE URINARY SYSTEM

PERSONAL IDENTITY DATA

Among the most important we have:

- Age.
- Sex.

Occupation.

Age

Acute diffuse glomerulonephritis often occurs in childhood and adolescence, in relation to the frequency of tonsillopharyngeal infections. Nephritic syndrome and congenital diseases such as polycystic kidney are also common.

Between the ages of 20 and 40, kidney and ureteral stones are most common, and are rarer after age 50.

Sex

In males, kidney stones, ureterovesical stones, and urinary obstructions due to prostatic hypertrophy are predominant; On the other hand, renal ptosis and pyelonephritis are highly frequent in women and are exacerbated during sexual life (honeymoon pyelitis) and pregnancy (gravid toxicosis, pyelonephritis, etc.).

In women, urinary tract infections are recurrent. Renal lithiasis due to infection is more common in women, while calcium lithiasis predominates in men.

Occupation

Harmful to the kidney and urinary tract are those activities in which the individual is exposed to heat and sweats a lot (which concentrates urine and predisposes to lithiasis) or those in which he handles nephrotoxic substances, such as mercury, lead, arsenic, hydroquinone-pyrogallol (photographic developer) and others. The frequency of lithiasis in sedentary professions, aviation pilots, etc. is known.

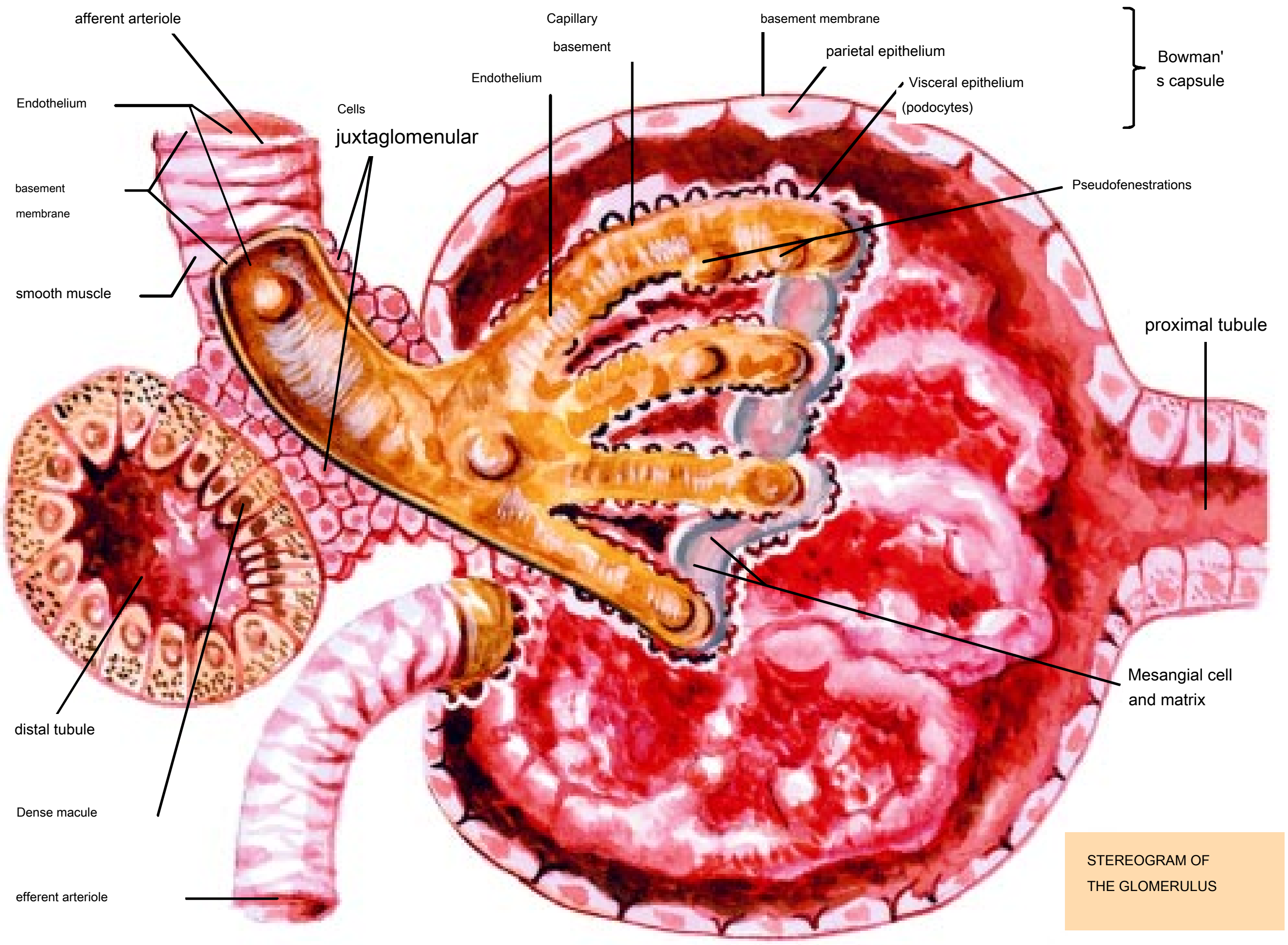
NEXT ANAMNESIS

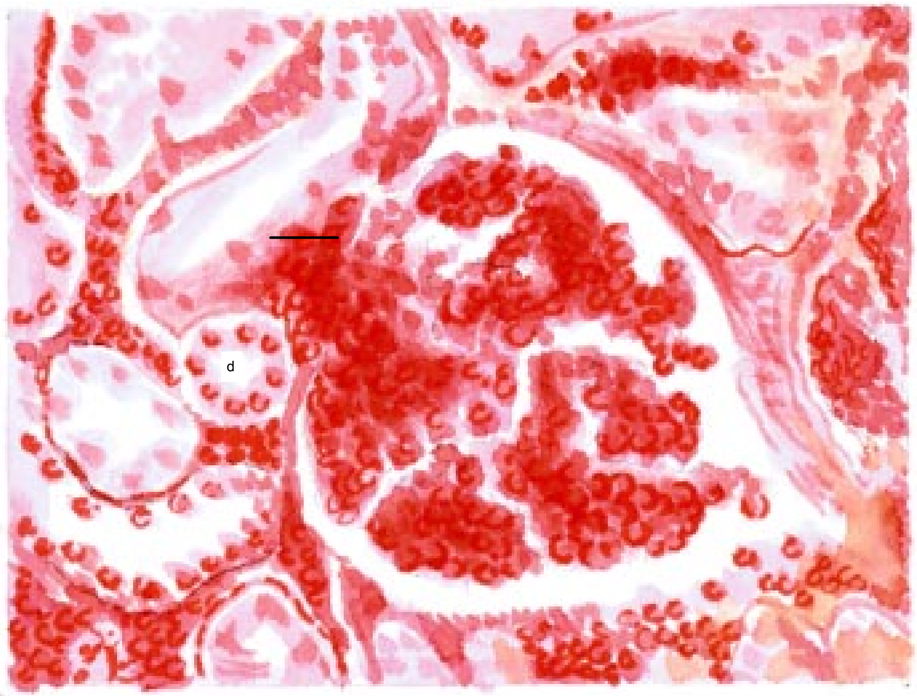
Start

It can be sudden, as happens in the pain of renal colic or in paroxysmal nocturnal hemoglobinuria, in which the individual, when dawn breaks, has red urine. Other times, however, the condition begins slowly, whether it is a sustained painful discomfort (lumbar or bladder) or disorders in urination (frequency, burning) that lead to a feverish condition with urinary tract infection (pyelonephritis).

---

*The antidiuretic hormone should be symbolized by the letters HAD, but the use of the English terminology antidiuretic hormone (ADH) has become widespread in textbooks, even in most of those written in Spanish, which is why - and in order not to create confusion for the student - we will sometimes use the acronym ADH, when referring to it.


afferent arteriole
Capillary
basement
basement membrane
parietal epithelium
Bowman'
s capsule
Endothelium
Visceral epithelium
(podocytes)
Endothelium
Cells
juxtaglomenular
basement
membrane
Pseudofenestrations
smooth muscle
proximal tubule
Mesangial cell
and matrix
distal tubule
Dense macule
efferent arteriole
STEREOGRAM OF
THE GLOMERULUS



Glomerulus (human):
Magnification X 350:
P: Proximal tubule
D: Distal tubule
J: Cells
juxtaglomerular

Fig. 66.4 Juxtaglomerular apparatus.

REMOTE Anamnesis

Previous illnesses

The most important are tonsillopharyngeal infections and scarlet fever, or the ingestion of nephrotoxic drugs (phenacetin and some antibiotics) in which a cause/effect relationship can be established.
However, in other kidney diseases no previous condition can be identified and the patient is consulted for a more or less advanced kidney lesion, which is considered primitive.

Chronic infections such as tuberculosis, osteomyelitis, etc., can lead to renal amyloidosis.

Transfusions of incompatible blood very frequently cause acute renal failure.

Diabetes and other metabolic diseases often cause serious kidney damage.

Trauma can cause kidney hematomas and hematuria.

MAIN SYMPTOMS AND SIGNS

The most frequent symptoms and signs of the urinary system are:

- Pain.
- Hematuria.
- Disorders in the evacuation of urine.
- Changes in urine color.
- Changes in urine density.

Pain

Pain of renal origin includes two modalities, depending on whether it originates in the capsule that covers the organ (simple or capsular pain) or in its excretory pathways (renal colic) .

Simple or capsular pain

Concept and semiogenesis

It is a pain, generally of low intensity, sitting in the lumbar region, which occurs when the renal capsule is irritated by an inflammatory or neoplastic process of the kidney or neighboring structures, or when it distends more or less abruptly due to a rapid increase in renal volume, as can occur in nephritis.

semiography

In pain, the semiographic elements listed in table 66.1 and discussed below must be taken into account:

1. Location. It is characteristic of the upper lumbar region. Lumbosacral pain rarely corresponds to the kidney. In some cases the healthy side hurts, contralateral pain.
2. Irradiation. The pain is frequently fixed, with little radiation. Sometimes, however, it radiates downward. Inverted irradiation, that is, from the hypogastrium to the flank or lumbar region, has great semiological significance when it occurs during urination, and can denote the existence of vesicoureteral reflux.
3. Intensity. Variable, depending on the cause of the pain, but generally its intensity is much less than that observed in renal colic.
4. Quality. Deep, dull, tenacious, sometimes with a constrictive character, other times lancinating.
5. Start mode. It usually sets in gradually, never with the suddenness of renal colic.
6. Way to calm down. It increases with walking and movements, and decreases with rest (if it is a stone lodged in the pelvis).
7. Duration. Variable, in relation to the causing entity; from hours and days in inflammatory processes, to months in tumor processes.
8. Symptoms and accompanying signs. Depending on the cause, different accompanying symptoms may occur, such as fever, cloudy urine, hematuria, and symptoms of bladder irritation (frequency, dysuria, urinary urgency).
On physical examination, the ureteropelvic points, as well as fist percussion, may be painful on palpation. Also, sometimes, an enlarged kidney can be palpated.

Table 66.1

Main semiographic characteristics of kidney pain
1. Location 2. Irradiation 3. Intensity 4. Quality 5. Start mode 6. Way to calm down 7. Duration 8. Symptoms and accompanying signs

Semidiagnosis

1. Acute pyelonephritis.
2. Intermittent hydronephrosis.
3. Kidney infarction:
   a) Embolic arterial obstruction.
   b) Atherosclerotic arterial obstruction.
4. Renal vein thrombosis.
5. Kidney trauma.
6. Polycystic disease with cyst rupture.
7. Hypernephroma (malignant neoplasm of the kidney).
8. Perinephritis and perinephritis hematoma.

Nephritic colic

Concept

It is a paroxysmal lumboabdominal pain syndrome produced by an acute pyeloureteral distension determined by an obstacle to the free flow of urine.

Semiogenesis

The starting point is the distension of the ureteropelvic cavities, determined by any obstacle to the free flow of urine. This distension stimulates the nerve endings, which carry the painful sensation through different afferent pathways, while producing reflexes that are manifested by spasms of the ureteral and pelvic smooth muscles; In this way, a vicious circle is closed that adds its effects.

The afferent pathways mentioned are always autonomic, vagal or sympathetic (figs. 66.5 and 66.6).

The stimulus is carried by these afferent pathways to the spinal cord, from where they can follow two paths:

1. Directly to the thalamus and from this, to the cortex, causing true visceral pain, which is characterized because the subject feels it deeply, located roughly in the real location of the viscera, without peripheral contracture or cutaneous hyperesthesia (area innervated by roots D8 to D10).
2. Upon reaching the posterior horn, the stimulus enhances the sensitivity of neighboring somatic neurons (Mackenzie) and through it reaches the cortex; It is referred pain, and the patient locates it in the skin areas corresponding to the sensory cerebrospinal nerves, stimulated at the level of the posterior horn through this mechanism (root areas D12 and LI).

Thus, the classic location of renal colic is the result of true visceral pain, and irradiation corresponds to pain referred to one or another of the spinal nerves corresponding to that spinal segment. If it irritates the genitocrural nerve, it reaches the testicle; If to the femoral skin, it is distributed along the external surface of the thigh; if to the shutter, on its internal face; and if you have greater sciatic pain, it is distributed from the buttock, along the back of the thigh and leg, and can reach the heel.

Since the source of irritation can also act on the motor neuron, muscle contracture can occur in the oblique, transverse, cremaster, etc. muscles.

semiography

In the pain of renal colic, the following must be taken into account:

1. Location. It is typically located in one of the lumbar regions (fig. 66.7). Sometimes, its location is anterior, in the hypochondrium, flank or iliac fossa of the affected side. In these cases it can be confused with other acute abdominal painful conditions, especially when accompanied by digestive symptoms.
2. Irradiation. From the lumbar region it radiates following the path of the ureter, downwards and forwards. Pain often extends to the testicle in men and the labia majora in women (see Fig. 66.7).
   Other characteristic radiations are towards the inner thigh and towards the anus. It can also radiate to the epigastrium and mesogastrium.
3. Intensity. It is stated that it is, in its most typical form, one of the most intense pains that a human being can suffer. The patient's reaction depends on their pain threshold and temperament. He is commonly prey to great agitation, adopting the most varied positions while sterilely searching for some relief. One characteristic is that the patient cannot remain immobile, a detail of great value to rule out peritoneal or root processes.
4. Quality. It is a continuous pain with violent paroxysms (colic) in relation to the contractions of the excretory duct, which determine an iterative sensation of expulsion ("the birth of the stone").
5. Start mode. It usually appears suddenly and immediately reaches its peak. Sometimes there are prodromal symptoms, such as lumbar or flank heaviness, hematuria, or frequency.
6. Duration. It is variable, from hours to a few days;
   This characteristic is greatly influenced by the therapeutic measures applied and the migration and eventual expulsion of the stone or clot.
7. Symptoms and accompanying signs. The most important- These are:
   - a) Urinary symptoms: hematuria, which can occur before, during or after colic, and oliguria, which, in exceptional cases, can lead to anuria.
   - b) Symptoms of bladder irritation: frequency, dysuria and bladder urgency.
   - c) Rectal symptoms: rectal pain and tenesmus.
   - d) Digestive symptoms: nausea and vomiting; sometimes paralytic ileus; Reflex abdominal defense may also be found.

Semidiagnosis

The cause of renal colic is represented by an obstacle of any nature in the excretory pathways of the kidney: stones, clots, lumps of pus, benign or malignant tumors, kinks and extrinsic compressions of various origins (tumors, inflammations, adhesions, anomalous vessels, retroperitoneal fibrosis, etc.). However, the most common cause of renal colic is ureteral lithiasis.

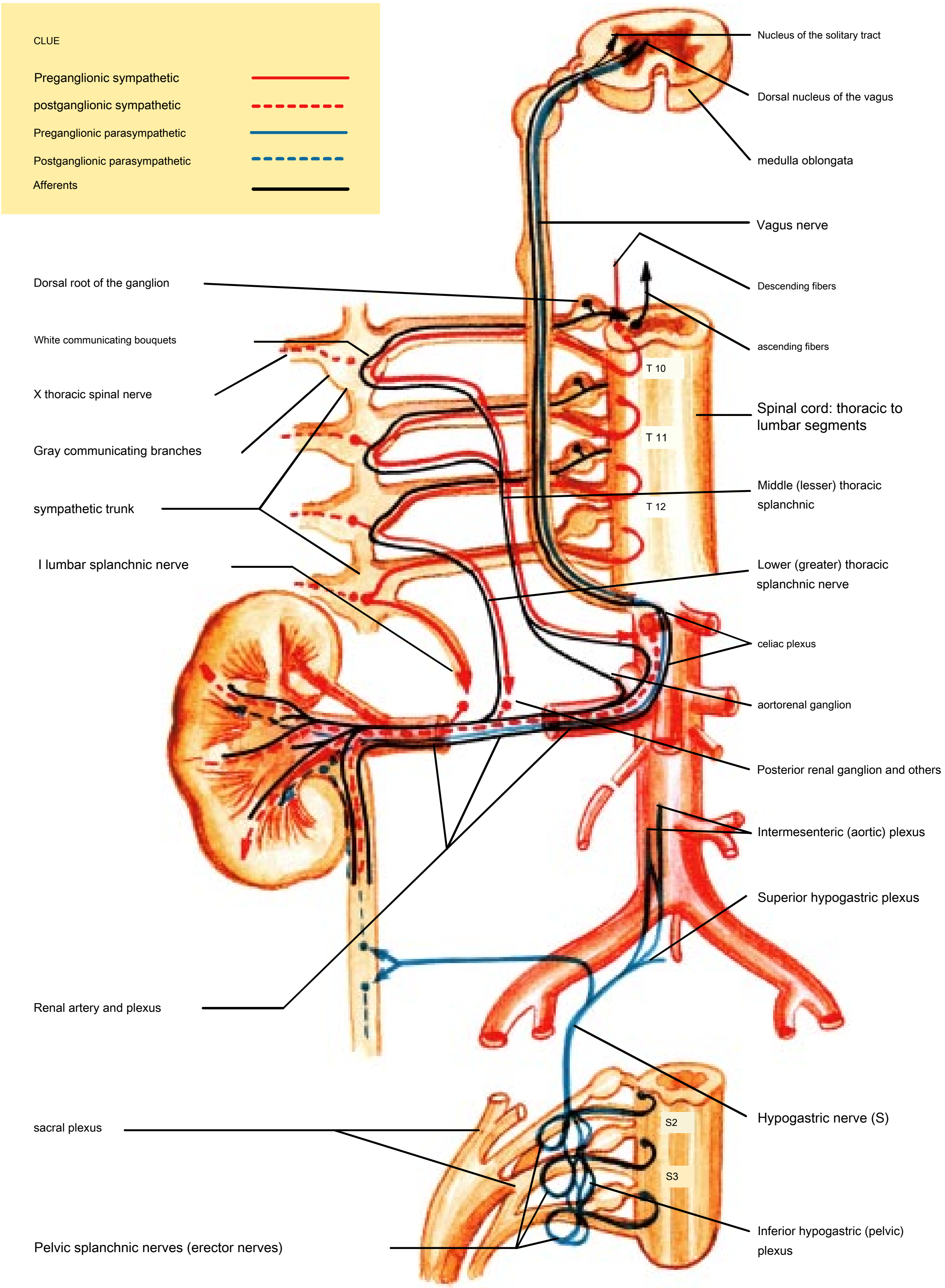

CLUE
Preganglionic sympathetic
postganglionic sympathetic
Preganglionic parasympathetic
Postganglionic parasympathetic
Afferents
Nucleus of the solitary tract
Dorsal nucleus of the vagus
medulla oblongata
Vagus nerve
Descending fibers
ascending fibers
Dorsal root of the ganglion
White communicating bouquets
X thoracic spinal nerve
Gray communicating branches
sympathetic trunk
I lumbar splanchnic nerve
T 10
T 11
T 12
Spinal cord: thoracic to lumbar segments
Middle (lesser) thoracic splanchnic
Lower (greater) thoracic splanchnic nerve
celiac plexus
aortorenal ganglion
Posterior renal ganglion and others
Intermesenteric (aortic) plexus
Superior hypogastric plexus
Renal artery and plexus
Hypogastric nerve (S)
sacral plexus
S2
S3
Pelvic splanchnic nerves (erector nerves)
Inferior hypogastric (pelvic) plexus

Fig. 66.5 Scheme of the innervation of the kidney and upper part of the ureter.

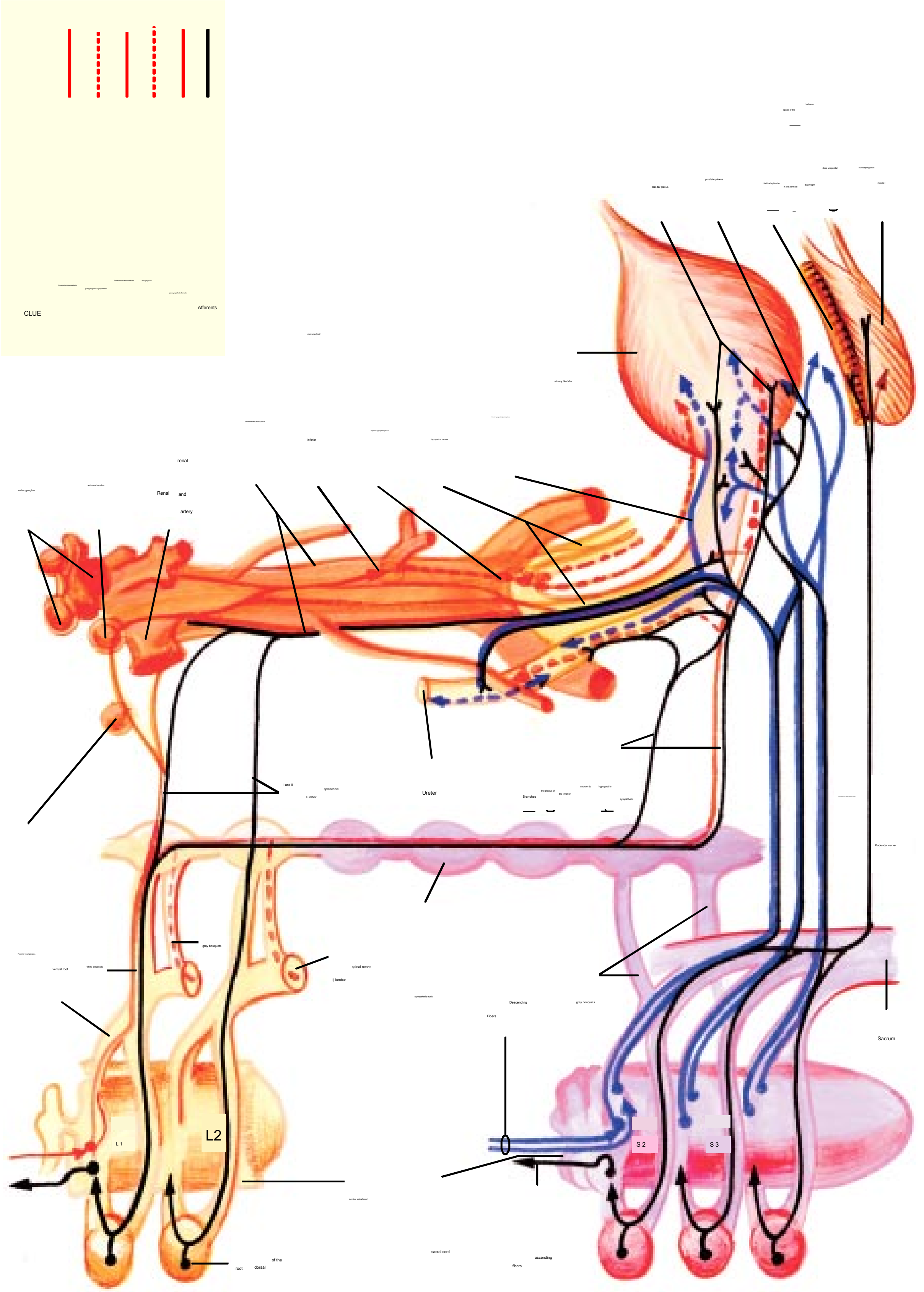

Preganglionic sympathetic
postganglionic sympathetic
Preganglionic parasympathetic
Postganglionic
parasympathetic Somatic
CLUE
Afferents
between
space of the
bladder plexus
prostate plexus
Urethral sphincter
in the perineal
diaphragm
deep urogenital
Bulbospongiosus
muscle (
mesenteric
urinary bladder
Intermesenteric (aortic) plexus
Superior hypogastric plexus
Inferior hypogastric (pelvic) plexus
inferior
hypogastric nerves
renal
celiac ganglion
aortorenal ganglion
Renal
and
artery
I and II
splanchnic
Lumbar
Ureter
Branches
the plexus of
the inferior
sacrum to
hypogastric
sympathetic
Pudendal nerve
Posterior renal ganglion
ventral root
white bouquets
gray bouquets
spinal nerve
I| lumbar
sympathetic trunk
Descending
Fibers
gray bouquets
Sacrum
L 1
L2
S 2
S 3
Lumbar spinal cord
of the
root
dorsal
sacral cord
ascending
fibers

Fig. 66.6 Scheme

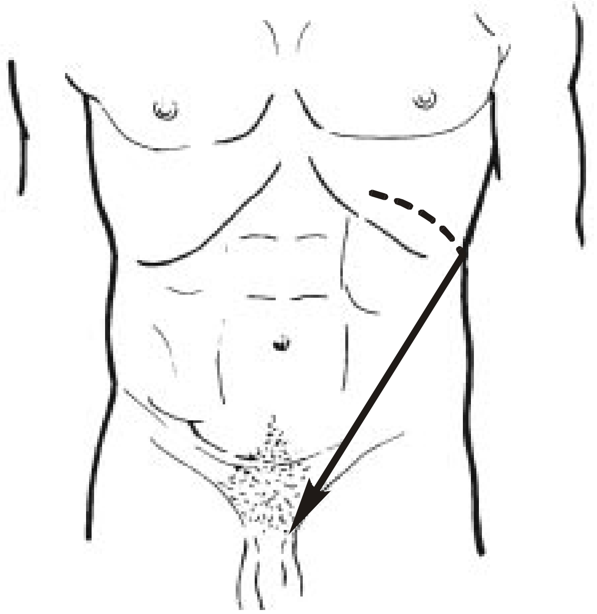


Fig. 66.7 Location and irradiation of renal colic.

Hematuria

Concept

Hematuria is the emission of bloody urine. When it is observable with the naked eye it is called macroscopic hematuria and when it is only identifiable with the microscope, microscopic hematuria. A few milliliters of blood can definitely stain a liter of urine.

Semiogenesis

Although hematuria commonly originates from an injury to the urinary tract, it is possible that it is caused by a general illness. The fundamental pathogenic mechanism is the lesions of the blood vessels (arteries, veins and capillaries), which allow blood to escape from its vascular bed.

Sometimes there is no anatomical lesion and hematuria is produced by local or general pathogenic changes. This includes hemodynamic and hemostatic alterations.

Semiotechnics and semiography

Identification of macroscopic hematuria is simple when it is present at the time we see the patient. Confusion with pseudohematuria due to blood coming, for example, from the uterus, or with hemoglobinuria, is ultimately resolved by urethral catheterization. In cases of doubt, direct microscopic examination of the urinary sediment will reveal the presence or absence of abundant erythrocytes.

The moment of urination at which hematuria occurs usually guides the approximate location of the point that bleeds Clinically it can be investigated using the classic three-glass test. The patient is made to collect the first part of the urination in one glass, continue it in a second glass, and use the third to finish urination.

If only the first denotes the presence of blood, or it is much more evident in it, we say that the hematuria is initial, and such a fact points towards bleeding coming from the urethra; In fact, the first urine carries the greatest amount of blood, and its subsequent accumulation is prevented by the continuous flow of urine. If it only appears in the third vessel, the hematuria is called terminal, and suggests that it is originating in the bladder itself. Logically, the blood has accumulated at the bottom of it and is mobilized at the end of urination when its contraction finishes emptying it. If all three vessels appear equally stained, we call it total hematuria, and the bleeding, although it can be located at any level, is usually renal or ureteral.

Microscopic examination of urine with phase contrast allows differentiation of glomerular hematuria from non-glomerular hematuria. Erythrocyte dysmorphism, basically of red blood cells with one or more extensions in their membrane (acanthocytes), as well as the presence of blood casts, locates the cause of hematuria at the glomerular level.

Semidiagnosis

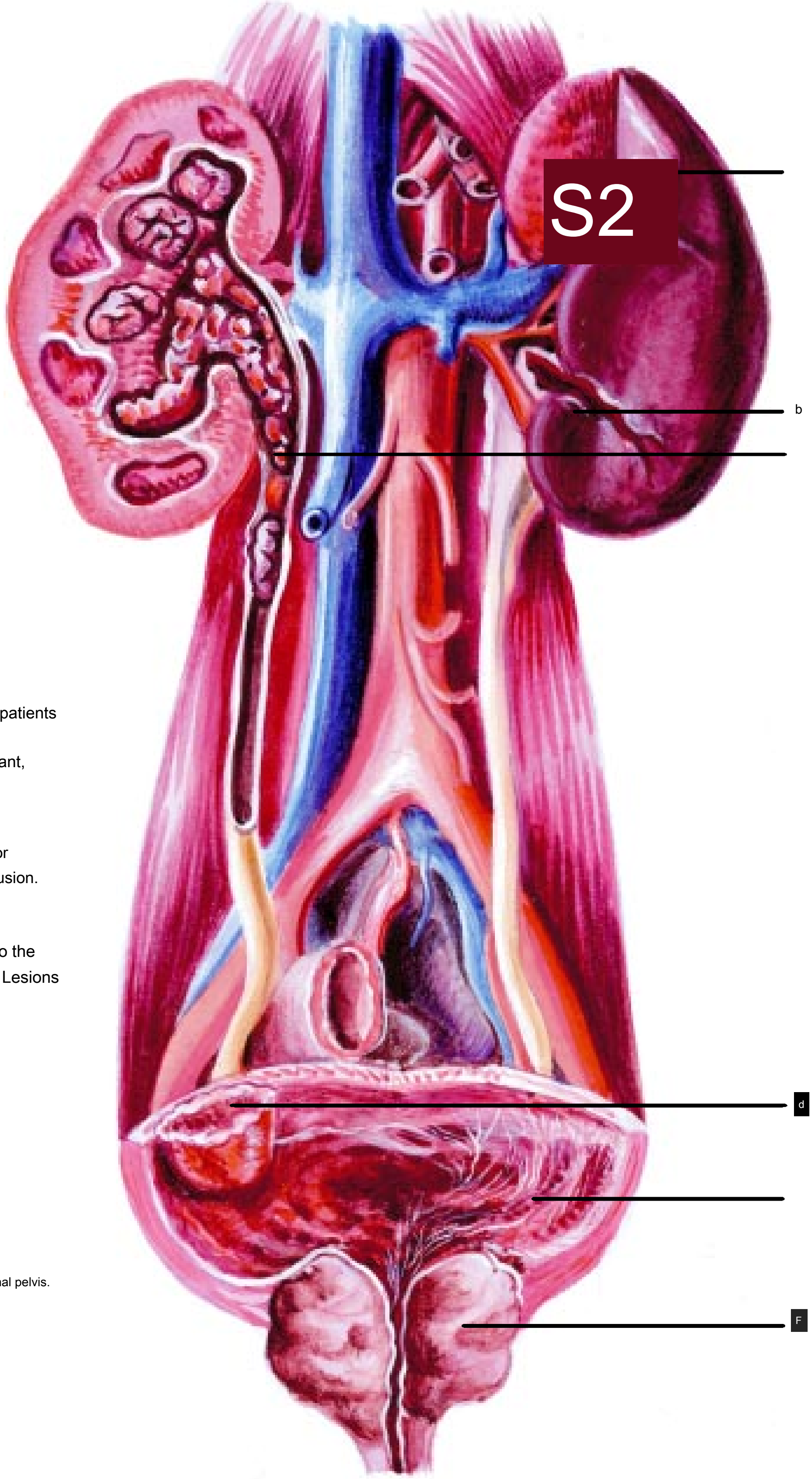
Hematuria is an alarm signal that always requires the most careful clinical evaluation. Blood can come from the kidney, upper excretory tract, bladder, prostate, and urethra. Hematuria of renal origin can be unilateral or bilateral.

The main causes of hematuria are (fig. 66.8):

1. Lithiasis (pyelocalyceal, ureteral, bladder, urethral).
2. Tumors (kidney, bladder, prostate).
3. Tuberculosis (of the kidney, ureter, bladder).
4. Of systemic origin (sickle cell trait, anticoagulant treatment, hemorrhagic diseases, particularly purpuric syndromes).
5. Other hematuria of renal origin (acute and chronic glomerulonephritis, renal infarction, polycystic kidneys, diabetic glomerulosclerosis, renal trauma).
6. Other hematuria of vesicourethral origin (hemorrhagic cystitis, bladder or urethral trauma, bladder polyps).

Urine evacuation disorders

They refer to the quantity (polyuria, oliguria, anuria), the rate of elimination (nocturia, opsiuria), or the form of urination (frequency when it is frequent, dysuria if it is difficult, etc.).



A: GLOMERULONEPHRITIS.

Some degree of hematuria is seen in all patients with acute glomerulonephritis; In chronic patients, hematuria, intermittent or constant, can persist for years.

B: TRAUMA.

Less violent trauma causes microscopic or macroscopic hematuria due to renal contusion. More violent trauma causes kidney lacerations that are accompanied by copious bleeding; If the lesion extends to the renal pelvis, blood appears in the urine. Lesions of the lower urinary tract also cause hematuria.

C: LITIASIS.

The presence of stones produces hematuria; he Renal colic is accompanied by hematuria macroscopic.

D: CARCINOMA.

In most cases of urinary tract cancer, hematuria is the first symptom; Tumors of the renal parenchyma do not produce hematuria as long as they do not compromise the renal pelvis.

E: CYSTITIS.

Acute cystitis is generally accompanied by terminal hematuria.

F: PROSTATE ADENOMA.

In prostate adenoma,

Fig. 66.8 Main causes of hematuria.

Polyuria

Concept

It is the increase in the total volume of urine in 24 hours above 2 L.

Semiogenesis

1. Hypotonic polyuria. The emission of low-density urine indicates a decrease in the facultative reabsorption of water at the level of the distal convoluted tubules and collecting ducts, determined by a deficiency of antidiuretic hormone (ADH) or by a resistance of these structures to the action of said hormone.

bow.

ADH deficiency is due to a temporary or permanent inhibition of the production or release of the hormone and may be related to different factors such as excessive fluid intake, reabsorption of overt or hidden edema, or hypothalamic-pituitary lesions (diabetes insipidus).

Tubular resistance to ADH can also be transient or permanent and constitutes the expression of a metabolic disorder (hypercalcemia, hypokalemia), hereditary disorder (nephrogenic diabetes insipidus) or due to chronic interstitial nephritis.

2. Osmotic polyuria. The emission of isotonic urine with

The plasma is an expression of an osmotic diuresis, determined by the elevation of the osmolarity of the plasma that offers a high load of solutes to the glomerular filtration, as occurs in hyperglycemia and hyperazotemia.

Semiodiagnosis (fig. 66.9)

1. Excessive ingestion of liquids (psychogenic polydipsia or potomania).
2. Decrease in tubular water reabsorption:
   a) Antidiuretic hormone deficiency. Central diabetes insipidus.
   b) With resistance to antidiuretic hormone. Hereditary nephrogenic diabetes insipidus.
   c) Diabetes insipidus acquired by:
      - Hypercalcemia.
      - Potassium depletion.
      - Obstructive nephropathy.
      Sjögren's syndrome.
      - Sicklemia.
      -Amyloidosis.
      Interstitial nephritis.
      Polyuric phase of acute tubular necrosis.
      - Drugs: amphotericin B, phenytoin, ethanol, demeclocycline.
3. Osmotic polyuria:
   a) Endogenous solutes:
      Diabetes mellitus (glucose).
      Chronic kidney failure (urea).
      Diuretics (NaCl).
      Polyuric phase of acute renal failure. (NaC1 and urea)
      .
      Kidney transplant (urea).
   b) Exogenous solutes:
      Mannitol.
      Hypertonic glucose.
      Saline solution.

Oliguria and anuria

Concepts

Oliguria is the decrease in the total volume of urine in 24 hours, below 500 mL (hourly urine output less than 20 mL).

Anuria is the extreme form of oliguria, with a urine output of less than 100 mL in 24 hours, which can reach zero.

Oligoanuria is a term that encompasses both stages of a single process characterized by low urinary output.

Semiogenesis

Prerenal Oligoauria

This syndrome is characterized by a reduction in glomerular filtration due to renal cortical hypoperfusion, which is immediately and completely reversed by restoring renal blood flow and is not associated with any structural damage to the kidney.

Renal oligoanuria

This syndrome is characterized by an acute reduction in renal function due to the perpetuation of renal hypoperfusion or the complete interruption of renal perfusion (medullary ischemia, cortical ischemia) or it may also be due to endogenous and exogenous toxic factors or renal damage caused by immunological factors.

In the transition from the prerenal to the renal stage, the mechanism of disseminated intravascular coagulation frequently operates at the level of the microcirculation, which aggravates the preexisting ischemia of the nephron and can cause acute tubular necrosis (postglomerular ischemia) and, in more advanced cases, bilateral cortical necrosis (preglomerular ischemia).

A: HYSTERICAL POLYURIA.

In hysteria, polyuria is due to the ingestion of large quantities of liquids.

B: DIABETES INSIPIDA.

Disorder of tubular reabsorption due to insufficiency of antidiuretic hormone results in the excretion of large amounts of very dilute urine.

C: HYPERALDOSTERONISM.

Potassium loss caused by an increase in aldosterone secretion impairs kidney function.

D: KIDNEY DISEASE.

In chronic diffuse glomerulonephritis and polycystic kidney disease, the kidney loses its ability to concentrate.

E: HYPERPARATHYROIDISM.

At the beginning of the disease, polyuria results from increased calcium and phosphorus; subsequently the deterioration of the kidney participates.

F: DIABETES MELLITUS.

In diabetes mellitus, polyuria is a direct result of glycosuria, because excessive glucose elimination requires an abnormal amount of water.

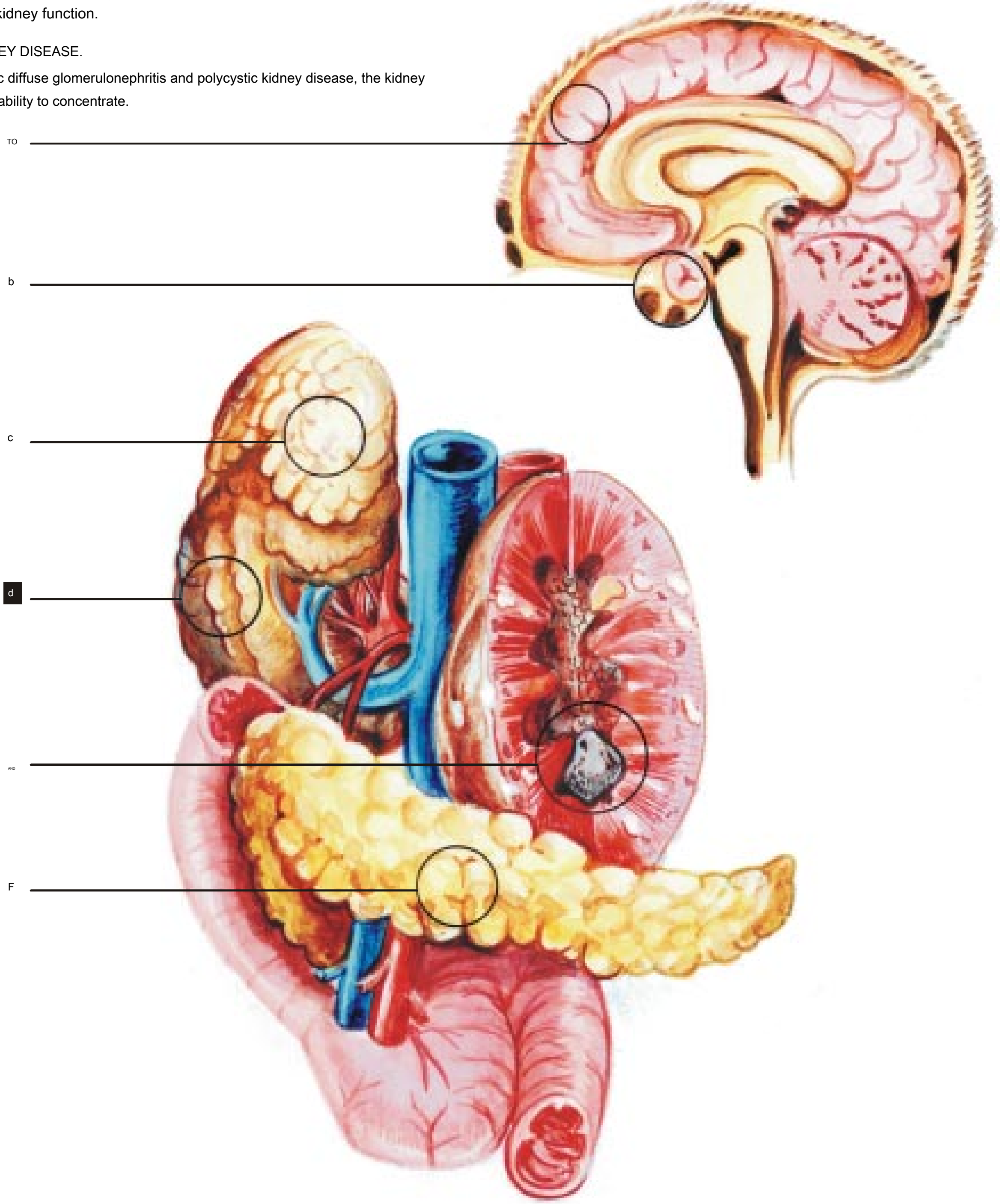


Fig. 66.9 Semidiagnosis of polyuria.

Here too, the urine volume is usually greatly reduced (15 mL/ h or less than 400 mL in 24 h), but shows the same osmolarity of the plasma and a high concentration of sodium and potassium as an expression of the failure of tubular reabsorption that is superimposed on the failure of filtration. Hyperkalemia is the most important electrolyte alteration of acute renal failure in the oligoanuric phase.

In renal oliguria, potassium is elevated for four reasons:

1. Accompanying acidosis.
2. Stress, which causes increased glycogenolysis.
3. Increased intracellular protein catabolism.
4. Decrease in excretion.

This elevation brings with it a serious danger to the patient's life if it is not adequately controlled.
As it is caused by an established injury, this oligoanuria persists for several days, until kidney function recovers. For the patient to survive until then, very strict monitoring and therapeutic control is required, with dialysis support.

Pathophysiological mechanism of oligoanuria in acute tubular necrosis

As Hamburger has pointed out, it might seem paradoxical that damage to the renal tubule, whose fundamental function consists of the reabsorption of primitive glomerular urine (glomerular filtration), was manifested by a decrease in urine volume and not by

Three mechanisms are known to explain this phenomenon.

less:

1. Total reabsorption of primitive glomerular urine by the damaged tubule, which has been expressed graphically by saying that the patient "urinates inside his kidney."
2. Mechanical obstruction by cylinders and various debris that hinder the passage of primitive urine through the injured tubular canaliculi.
3. Glomerular insufficiency resulting from ischemia of the glomerulus, with marked reduction in filtration.

In fact, the three mechanisms mentioned are associated, each participating, with greater or lesser intensity, in the production of oligoanuria.

Postrenal oligoanuria

It is caused by intrinsic or extrinsic obstruction of the upper excretory tracts (above the bladder). The obstruction can be bilateral or unilateral; In the latter case it is due to the fact that the opposite kidney was previously damaged (non-functional).

semiography

Oligoanuria is a complication of another usually serious and obvious disorder. Requires exact measurements of urine output, hourly and 24 hours.

It should not be confused with urinary retention due to prostatic or urethral obstruction, conditions in which a small volume of urine can also be eliminated.
In these cases, examination of the lower abdomen reveals the presence of a bladder full of urine or bladder balloon - which appears as a central tumor located in the hypogastrium and which in extreme cases can reach the navel -, rounded, with a smooth surface, resistant to palpation and dull to percussion. Bladder catheterization obtains a large amount of urine and makes the "tumor" disappear.

Semidiagnosis

1. Prerenal oligoanuria:
    a) Absolute or relative depletion of blood volume due to hemorrhages, dehydration due to renal and extrarenal causes, and extensive burns.
    b) Relative volume depletion, as seen in liver cirrhosis and nephrotic syndrome.
    c) Reduction in cardiac output: heart failure, extensive pulmonary embolism and low output syndromes.
    d) Intense peripheral vasodilation: antihypertensive medications, anesthesia, drug overdose.
    e) Loss of renal autoregulation: elderly people who take prostaglandin synthesis inhibitors and in patients with previous nephropathies.
2. Renal oligoanuria:
    a) At the level of the renal interstitium, in acute interstitial nephritis caused directly or indirectly by staphylococci, gram-negative germs, brucellosis, fungi, viruses, etc.
    b) At the glomerular level, in rapidly progressive (crescentic) glomerulonephritis, due to acute post-streptococcal glomerulonephritis and systemic diseases.
    c) At the level of small intrarenal vessels: uremic-hemolytic syndrome, pregnancy toxemia, disseminated intravascular coagulation, etc.
    d) Acute tubular necrosis, due to medullary or cortical ischemia, or due to exogenous or endogenous toxins.
    Exogenous toxins are: antibiotics (aminoglycosides, sulfas, etc.), anesthetic agents, radiological contrast media, analgesics, anti-ulcers, diuretics, chemotherapy, immunosuppressants, organic solvents, heavy metals, insecticides, herbicides, amphetamines.

The endogenous toxicants that produce acute tubular necrosis are pigments such as myoglobin and hemoglobin.

3. Postrenal oligoanuria:

a) Intrinsic obstruction (ureteral stones).

b) Extrinsic compression (malignant tumor, retroperitoneal fibrosis, among others).

Nocturia

It is the reversal of the normal rhythm of urine elimination; during the night it is equal, and even greater, than during the day. Its semiological value is very broad, both in renal and extrarenal conditions: acute or chronic nephritis, polycystic kidney, congestive heart failure, prostate conditions, portal hypertension, presence of edema of any etiology, etc.

Opsyuria

This is the name given to the delay in the elimination of ingested water. If a healthy person, with no previous water deficit, drinks a liter of water in half an hour, after two hours he will have eliminated three quarters and the rest two hours later. This does not occur in patients with heart or kidney failure and in cirrhotics, in whom only a small part of the water passes into the urine within these hours; This fraction is still scarce if the patient is up and walking.

Frequencyuria

We say that there is frequency when urination is very frequent, but in very small quantities, so that the 24-h diuresis may be normal or slightly altered. It is very often accompanied by urinary urgency and other urination disorders. It is mainly observed in conditions of the bladder, prostate and urethra. It is also common during pregnancy.

Dysuria

Dysuria is known as the difficulty in performing the act of urination, determined by some obstacle to the normal flow of urine from the bladder to the outside, or by any cause that causes an impediment to good bladder contraction.

Dysuria occurs in some neurological conditions or in diseases of the prostate, bladder, and urethra.

Drugs that block parasympathetic impulses (stimulators of the longitudinal fibers of the urinary bladder, detrusor urinae, and inhibitors of the internal sphincter), that stimulate the sympathetic (opposite action to the previous one) or that block the ganglia of the vegetative system, also produce dysuria.

Dysuria can present in different forms:

Slow urination. When the act of urination lasts longer than normal.

Delayed urination. When the time is prolonged, from the voluntary beginning of the act of urination to the beginning of the emission of urine. It usually requires effort on the part of the patient.

Abrupt interruption of the jet. As sometimes occurs in bladder stones or pedunculated tumors of that organ, they are dragged by the flow of urine towards the urethral orifice, abruptly occluding it.

Although dysuria means difficulty or pain when urinating, in women, its best translation is burning when urinating. This symptom is very common in women with vulvovaginal, urethral and bladder infections.

Dysuria at the beginning of urination, and even without urinating, reflects a vulvovaginal infection rather than a urinary tract infection.

Dysuria throughout urination localizes the infection in the urethra, and dysuria at the end of urination, accompanied by urinary urgency, is due to a bladder infection (cystitis). In cystitis there is also daytime and nighttime frequency. If nocturnal frequency is absent, the infection is most likely confined to the urethra only.

Bladder urgency

It is the continuous, painful and ineffective desire to urinate without urination causing the discomfort to subside. Very little urine is expelled with each urination, and sometimes none at all. Its causes lie in the lower urinary tract or the bladder, but tenesmus can also be due to neurological processes or be produced by certain drugs.

Overflow urination

Urine flows drop by drop through the urethral meatus. It is observed in cases of atonic bladder, distended by a prostatic adenoma and coexists with a bladder balloon.

Urine incontinence

It is the inability of the bladder to retain urine.

It must be differentiated from overflow urination, described above. It is estimated that around 5% of adult women suffer from it and it is caused by exertion (coughing, sneezing and others), by a deficiency in the urethral sphincter mechanism, by neurological disorders, etc.

Changes in urine color

The usual color of urine varies between pale yellow and amber: these oscillations are related to the urinary volume and consequently, the concentration of urochrome, which is the most important pigment in the normal color of urine.

The darkest physiological urine is highly concentrated acidic urine, which shortly after being emitted becomes dark due to the oxidation of the pigments.

The color of urine may vary according to your pathological conditions:

1. Reddish brown. In febrile states, due to its greater concentration and abundance of urates and uroerythrin.

2. Intense yellow, mahogany color. With a certain greenish tint at times, in the different types of jaundice, due to bile pigments and excess urobilin.
3. Reddish 0 red. In hematuria and hemoglobinuria. If the urine is acidic, hemoglobin can be transformed into methemoglobin and hematin, taking on a brown color.

4. Brownish. In melanotic sarcomas, due to the transformation of melanogen into melanin upon oxidation. The same coloration can be observed in alkaptonuria, in urine several hours after being emitted. Paroxysmal nocturnal hemoglobinuria is characterized by the emission of blackish-brown urine during the first urination in the morning.

5. Violet red. As seen in rare cases of porphyrinuria.

6. Yellowish, milky white. In chylurias, and more rarely, in very intense pyurias. Chyluria is the presence of lymph in the urine and is due to the obstruction of the lymphatic ducts in the upper abdomen, with rupture of the small lymphatics of the renal pelvis. The causes are: filariasis, abdominal and gynecological tumors and aortic aneurysm.

7. Various (greenish, reddish, brown, etc.). Due to the presence of medications, such as mercurochrome, methylene blue, guaiacol and many others, or due to the elimination of dyes used in food preparation.

Changes in urine density

When diuresis remains normal, between 800 and 1,500 mL, the density of urine usually varies between different voids, between 1,015 and 1,025.

Physiologically, 1density is in inverse relationship with the volume of urine eliminated. In large polyurias, due to excess fluid ingestion, for example, urine usually has a lower density and in marked oligurias, such as dehydration and low fluid ingestion, it reaches maximum density values. Since the density is related to the quantity amount of solid substances dissolved in the urine, we must realize, to give it its fair value, that there is no significant albuminuria or glycosuria. It is an index of satisfactory renal function that the density of urine oscillates only in accordance with physiological variations, and clinically we assume good renal function when we observe high-density concentrated urine (for example, 1,030), assuming that there is no marked albuminuria or glycosuria.

This ability of the kidney to concentrate urine, producing diuresis of normal density, is called sthenuria.

When the density is pathologically decreased, we say that there is hyposthenuria, and we see low-density urine. Hyposthenuria occurs in certain stages of renal failure, consecutive to different nephropathies: chronic diffuse glomerulonephritis, polycystic kidney, etc.

At this stage, the kidney can maintain a normal diuresis, which is known as pseudonormaluria, or in more advanced stages, produce compensatory polyuria, in an effort to rid the body of catabolites that would be harmful if retained.

Sen.

If renal failure is more marked, not only is there low density, but in practically all urinations the urine contains equal volume and equal density; These phenomena are known by the names isovolume and isosthenuria, respectively.

We will not insist on broader details, since the study of density will be explained in the Clinical Laboratory subject, with all the extension required.

For the same reason we omitted proteinuria, cylindruria and other urinary alterations, which are of great interest in the diagnosis of nephropathies.

ALTERATIONS TO THE PHYSICAL EXAMINATION

Inspection

Although it provides little data, sometimes inspection can reveal the presence of tumors in the hypochondria and flanks, expression of unilateral or bilateral renal enlargement. Inflammatory signs or hemorrhagic suffusions indicative of a perinephritic process (abscess, hematoma) may appear in the lumbar fossa.

Palpation

Normally the kidneys are not palpable and the classic maneuvers, useful to demonstrate this physiological condition or the discrete growth or decrease thereof, were already described in volume 1, Section I, Chapter 13. In the clinic, large renal tumors are diagnosed by classic abdominal palpation, and raise the differential diagnosis of a tumor in the right or left hemiabdomen.

Enlargement of the kidney (nephromegaly)

Under normal conditions, in thin people, the lower pole of the right kidney could be palpated). When a kidney can be palpated, it usually indicates the existence of an abnormal position (ptosis, ectopia) or an enlargement of the viscus. Here, we will only study this last eventuality.

Semiogenesis and semidiagnosis (fig. 66.10) 1.

Compensatory hypertrophy. In cases of congenital or acquired solitary kidney (functional or surgical ablation of the other kidney).

2. Pyelocalyceal distention due to obstruction of the excretory duct. Hydronephrosis or pyonephrosis (if associated with suppuration), generally caused by stones; These can be found in the kidney, ureter and bladder, and occasionally, in the urethra and prostate; They vary greatly in size, shape and chemical composition and when they do not cause severe urinary obstruction, no simple sign can be considered pathognomonic, but the usual symptoms are: localized, dull and constant pain or radiating pain (renal colic), which spreads in the direction of the ureter to the bladder; hematuria and pyuria.
3. Perinephric swelling. Perinephric hematoma (traumatic), cellulitis and perirenal abscess.
4. Nephrosis. It is an injury that mainly affects the renal tubules. Among the clinical signs are massive edema and albuminuria.
5. Cystic enlargement. A single cyst or multiple cysts can often reach enormous size and occupy almost half of the abdominal cavity; They can destroy most of the kidney substance. The polycystic kidney is enlarged and presents symptoms of renal failure, hematuria, and often hypertension and progressive anemia.
6. Tumor enlargement. Kidney tumors originate in the parenchyma or epithelium that lines the pelvis and calyces. They are generally malignant. In adults, the most common parenchymal lesion is hypernephroma or carcinoma, while in children the most common tumor is embryoma (Wilm's tumor).

Renal ureteral pain points

The ureteropelvic points, especially the posterior ones (see Fig. 18.5, in volume 1), are usually painful in pyelonephritis and renoureteral lithiasis, among other entities.

Presence of edema

Edema is an important sign in renal semiology, which is detected by inspection and palpation. It has already been studied in volume 1, Chapter 26 and will also be analyzed in the following chapter, on renal syndromes.

Percussion

Lumbar fist percussion is intolerable in perinephritis, as it is a pain of capsular origin.

Auscultation

The finding of a systolic murmur in the lumbar regions and flanks suggests the existence of partial stenosis of the renal artery, which is the cause of renovascular hypertension. Renal arteriovenous fistula, renal artery aneurysm, and splenic aneurysm (left 1st side) also produce murmurs at this level.

COMPLEMENTARY EXAMS

The clinical diagnosis, that is, the one made exclusively with the data provided by the interrogation and the physical examination, constitutes the primary and guiding element in the management of the patient, but the so-called auxiliary diagnostic methods sometimes play an important role, which is why they must be well known by the doctor. Here we will make a brief explanation of these, since they will be considered extensively in the subject of Urology.

The complementary exams include:

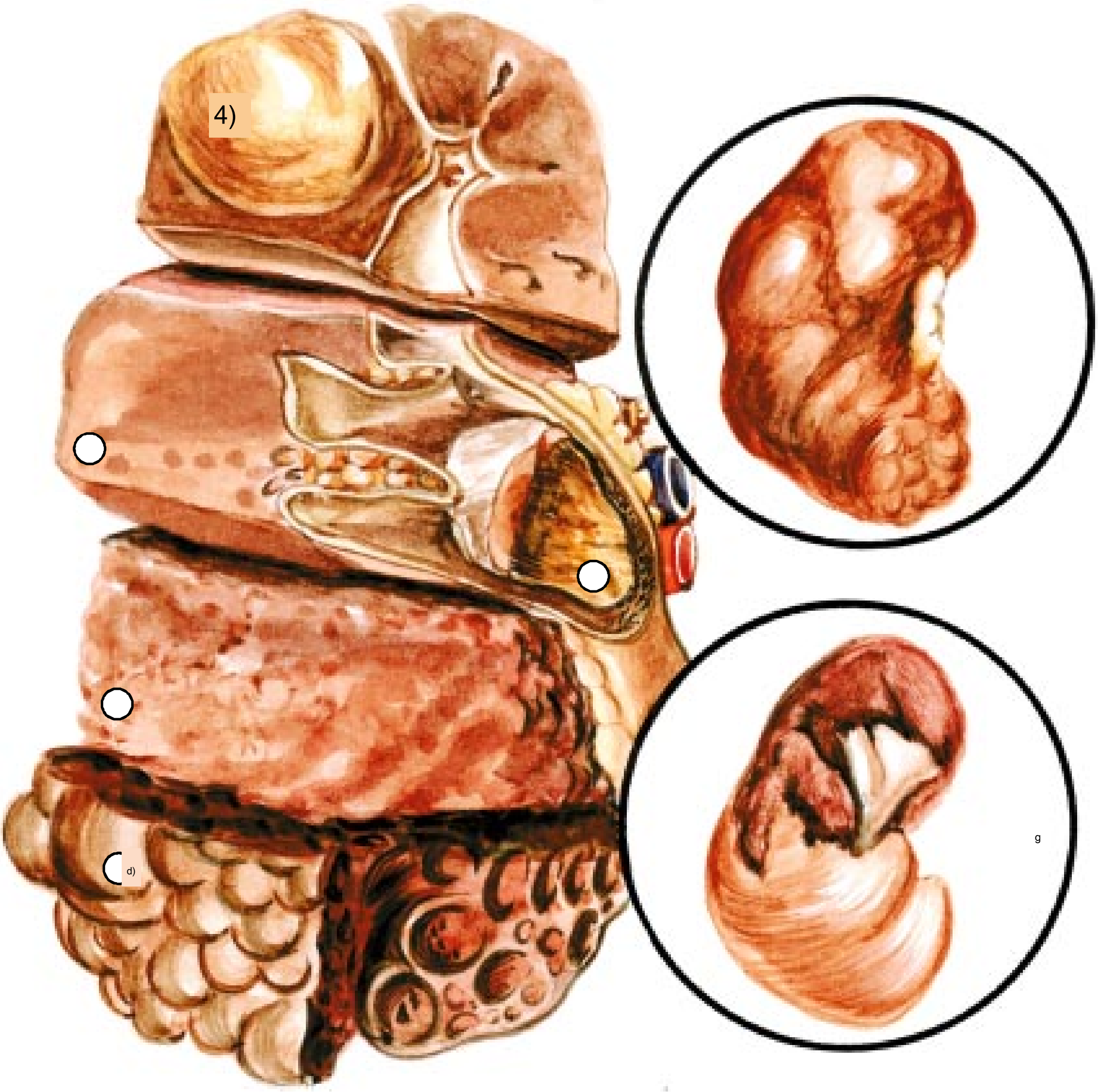
Laboratory exams.

- Endoscopic examinations.
- Imaging examinations
- Histological examinations through biopsies.

LABORATORY EXAMS

General urine examination. Quantity, color, appearance, density, reaction, chemical and sediment examination.

Cituria. Urine obtained at random by sterile urination (medium stream) not centrifuged. Proteinuria should be determined by the 20% sulfosalicylic method (results of the qualitative method: negative, one cross, two crosses and three crosses).



A: TUMORS.

Kidney tumors originate in the parenchyma or in the epithelium that lines the pelvis and calyces. They are usually malignant. In adults, the most common parenchymal lesion is hypernephroma or carcinoma, while in children the most common tumor is embryoma (Wilm's Tumor).

B: NEPHROSIS.

It is an injury that mainly affects the renal tubules.

C: VASCULAR DISEASES OF THE KIDNEY.

They can be classified into two groups: disease of the renal arterioles (benign hypertension produces renal sclerosis and, sometimes, mild renal failure, and malignant hypertension produces intense renal sclerosis and marked renal failure) and senile arteriosclerosis (the kidney is contracted and with numerous scars, as a result of infarctions caused by atheromatous modifications of the aorta and renal artery).

D: CYSTIC KIDNEY.

A single cyst or multiple cysts can often reach enormous size and occupy almost half of the abdominal cavity. They can destroy the most of the kidney substance. The polycystic kidney is enlarged and presents symptoms of renal failure, hematuria and often hypertension and progressive anemia.

E: CALCULATIONS.

They can be found in the kidney, ureter and bladder, and occasionally in the urethra and prostate. They vary greatly in size, shape and chemical composition. When they do not cause severe urinary obstruction, no simple sign can be considered pathognomonic, but the usual symptoms are: localized, dull and constant pain or radiating pain (renal colic) that spreads requiring the direction of the ureter to the bladder, hematuria and pyuria.

F: GLOMERULONEPHRITIS.

It is an inflammation of the glomeruli, but can produce secondary injury to the renal tubules at a later stage; It occurs in the following forms: acute (characterized by the presence of casts and red blood cells in the urine, renal failure), subacute (characterized by edema and albuminuria) and chronic (characterized by hypertension and renal failure).

G: TRAUMA.

Fig. 66.10 Semidiagnosis of nephromegaly.

Urinary sediment counting is performed in a erythrocyte counting chamber (Neubauer).

Urinary sediment count Normal figures

10,000 leukocytes/mL (10x 10® L) 10,000
red blood cells/mL (10x 10® L) No cylinders

Addis Count

Normal figures

Proteins: < 0.03 mg/min
Red blood cells and leukocytes: < 1,000/min
Cylinders: < 250/min

Addis count. Cell and cylinder counts of centrifuged urine. The patient is placed on a liquid-restrictive diet. Timed urine collection, 2 h, 4 6 24 h. Determination of minute proteinuria by the Biuret method.

Blood chemistry. Blood determinations of creatinine, urea, uric acid, non-protein nitrogen, other catabolites and electrolytes.

Kidney function tests (glomerular filtration rate). They consist of the study of the way some substances are eliminated by the kidney, by comparing their content in the blood with the amount eliminated through the urine, for which it is essential to know the substance and the amount administered.

The most used method in clinical practice is the determination of plasma creatinine and its renal clearance. The ideal marker to measure glomerular filtration rate is inulin by the continuous infusion method and spontaneous urination. Three urinary clearances are performed per patient and the average inulin clearance is determined.

There are other alternative methods that are much easier to perform, faster and in all respects similar to the clearance of inulin, such as the clearance of Tc, DTPA and I125 iodothalamate.

The dilution and concentration test can also be considered as a kidney function test.

ENDOSCOPIC EXAMINATIONS

They are performed using optical instruments that allow visualization of the urethra and bladder (panendoscopy), the bladder only (cytoscopy), or the ureter and renal pelvis. Ureteroscopy and pelvicoscopy are performed through percutaneous nephrostomy, and even the renal calyces can be visualized.

IMAGING EXAMINATIONS

These methods are used in three types of situations:

1. As screening methods for the rapid diagnosis of urinary tract dilation, skeletal abnormalities or tumors.
2. To determine the site and cause of the obstruction.
3. To specify renal and retroperitoneal tumors.

Simple x-ray or simple urinary tract (TUS)

The simple urinary tract is a radiological examination of the abdomen, without contrast but with prior preparation, which provides guidance on the shape and size of the renal shadow, the presence of a megabladder in lower obstructions, radiopaque stones, calcifications of the renal parenchyma, as well as a calcified aortic aneurysm and calcifications of gynecological tumors. Furthermore, it specifies the contours of the psoas muscles and reveals skeletal abnormalities in myelomeningocele and bone metastases in prostate carcinoma.

abdominal ultrasound

Ultrasound or abdominal ultrasound allows us to visualize the shape, size and location of the kidneys, the presence of dilations or obstructions of the urinary tract and the presence of renal and extrarenal tumors.

Excretory urography or descending urogram

Elimination through the urinary tract of the radiopaque contrast material, injected through the vein. It allows visualization of the upper urinary tract, especially the apparatus excretory

Excretory urography allows us to see, from the nephrographic phase, kidney function separately, and in successive views to be able to show the site of an obstruction and, in many cases, the cause of it.

Ascending pyelography

Direct introduction of contrast into the ureters and pelvirrenal cavities. It is necessary to catheterize the ureters, after cystoscopy.

When kidney function is compromised, unilaterally or bilaterally, it is necessary to complement it with retrograde or antegrade pyeloureterography.

Cystography and urethrocystography

They can constitute the final phase of the descending urogram, if the doctor so wishes, but they are generally indicated as individual studies, when only the investigate the bladder and urethra. It consists of the introduction of radiopaque contrast into the bladder by means of a probe. Views are taken of the full bladder and, after removing the catheter, while the patient urinates.

Retropneumoperitoneum

Injection of air or oxygen into the retroperitoneal space. The renal profile is better defined.

Aorthography

Pressure introduction of iodinated contrast (at 759%) into the aorta, after puncture through the left lumbar region or catheterization of the aorta. The renal arteries are well visualized.

Cavography

Contrast in the lower cava. It is used to look for tumor thrombi in cases of kidney tumor.

Retropneumoperitoneum, aortography and cavography, in general, have been displaced by more modern and less invasive imaging methods, which we will point out below.

Computed axial tomography (CAT)

It has value in revealing the etiology of retroperitoneal processes (retroperitoneal fibrosis, tumors, etc.).

Nuclear magnetic resonance (MRI)

It gives a very good anatomical and functional definition, it does not require radiological contrast media and its fundamental indication is retroperitoneal pathologies.

Scintigraphy

It uses isotopes and allows, with its fixation in the renal parenchyma, the study through images of its lesions.

Static (Tc DMSA) or dynamic (Tc MAG3) renal scintigraphy allows exact measurement of the renal function of each kidney and, in urological diseases, separates dilation from urinary tract obstruction.

HISTOLOGICAL EXAMINATIONS THROUGH BIOPSIES

We will only point out the biopsies (kidney, bladder, prostate). Kidney biopsy is of great importance in the diagnosis of some nephropathies.

MAIN SYNDROMES OF THE URINARY SYSTEM

67

Introduction

In this chapter we will study the most frequent syndromes that occur in conditions of the urinary system:

- Acute renal failure syndrome.
- Chronic kidney failure syndrome.
- Nephrotic syndrome.

ACUTE RENAL FAILURE SYNDROMVE

CONCEPT

Acute renal failure is a clinicohumoral syndrome resulting from the sudden suppression of renal function. It usually causes profound homeostatic disturbances, such as the retention of waste products and hydroelectrolyte imbalances, which seriously endanger the patient's life. Oligoanuria, although common and characteristic, may be absent (non-oliguric acute renal failure or high urinary output).

SYNDROMOGENESIS0 PATHOPHYSIOLOGY

The mechanisms that cause acute renal failure (prerenal, renal, and postrenal) have been widely considered in the semiogenesis of oligoanuria (see Chapter 66).

SYNDROMOGRAPHY 0 POSITIVE DIAGNOSIS (FIG. 67.1)

clinical picture

Background

Acute renal failure is always a complication in the course of a pre-existing morbid process. Its timely identification depends largely on the degree to which it is suspected in patients with any of the predisposing disorders indicated (see oligoanuria, Chapter 66).

Symptoms

Apart from those inherent to the causative disease, acute renal failure causes symptoms related to the retention of waste products and to the disturbances of hydroelectrolytes and acid-base balance that progressively take hold. Decreased diuresis is a characteristic and frequent sign, although not obligatory.

Early diagnosis of this complication requires monitoring of hourly diuresis and blood urea and creatinine concentrations in all predisposed patients.

Depending on the etiology and the opportunity for diagnosis and treatment, different hydromineral imbalance syndromes may be found, such as dehydration or overhydration and water intoxication, sodium depletion or excess, hyperkalemia and metabolic acidosis, all of which will be studied in Chapter 69.

With the progression of kidney failure, clinical manifestations appear in different organic systems, similar to those that we will describe in the terminal stage of chronic kidney failure and which are part of the uremic syndrome.

Acute oliguric renal failures that survive pass into the post-oliguric or diuretic phase, characterized by ineffective polyuria since it alone does not adequately resolve nitrogen retention or hydroelectrolyte disorders. Strictly speaking, the latter could even worsen if they are not properly attended to and controlled by the doctor.

Complementary exams

Urine. Through this test, the density, osmolarity, and concentration of sodium and sediment are determined. The values of urine examination parameters, which differentiate prerenal from renal acute renal failure, are shown in Table 67.1.

Blood. In a blood test we can determine if urea, creatinine and uric acid are elevated; in it ionogram shows alterations in sodium (normal, high or low) and potassium (severe hyperkalemia, which can sometimes cause death);
and with blood pH and hemogasometry, metabolic acidosis.

Although the concentration of urea in the blood is the most commonly used clinical index to estimate renal function, it is not the most accurate, as it is strongly influenced by extrarenal factors, such as hypovolenemia and protein intake. Freed from these influences, the concentration of creatinine in the blood reflects kidney function more closely.

ECG. Together with the ionogram, it allows monitoring the appearance of electrolyte disturbances, in particular hyperkalemia. It can also detect associated pericarditis.

ETIOLOGY

The causes have been exposed when studying the semidiagnosis of oligoanuria (Chapter 66).

CHRONIC KIDNEY FAILURE SYNDROMIE

CONCEPT

Chronic renal failure is a clinicohumoral syndrome resulting from the sustained and progressive loss of renal function; This syndrome can vary from the subclinical stage to the florid symptomatic stage, and whose final destination is uremia.

In its final phase it is fatal in more or less long periods, unless a treatment can be performed that replaces kidney function through dialysis and/or kidney transplant.

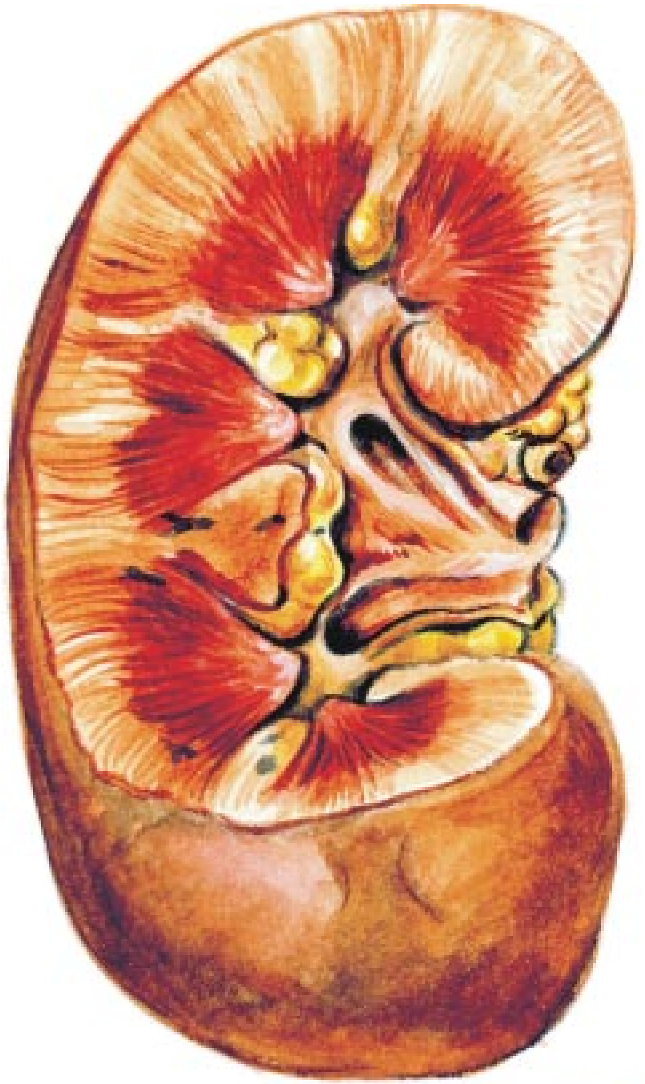
SYNDROMOGENESIS PATHOPHYSIOLOGY

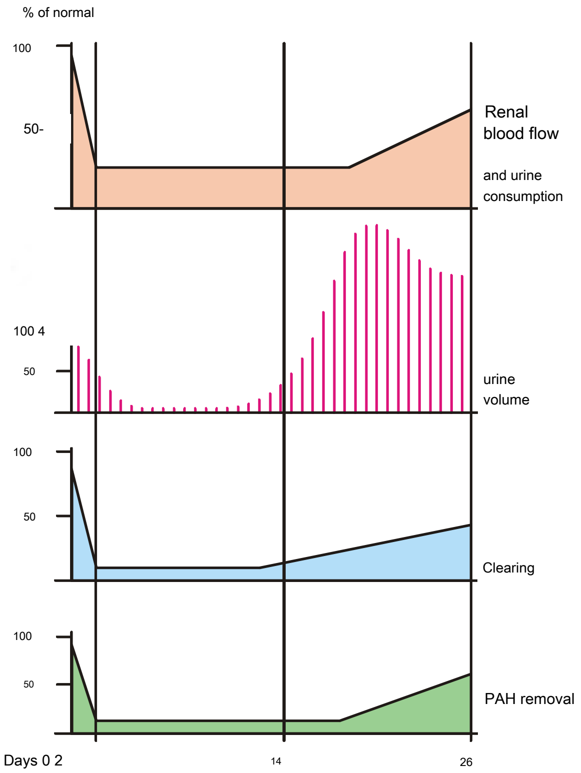
For a nephropathy to give rise to this syndrome, it must have two fundamental characteristics: be bilateral and diffuse, and have a slow and progressive evolution.

Table 67.1

Values that distinguish prerenal acute renal failure from renal failure

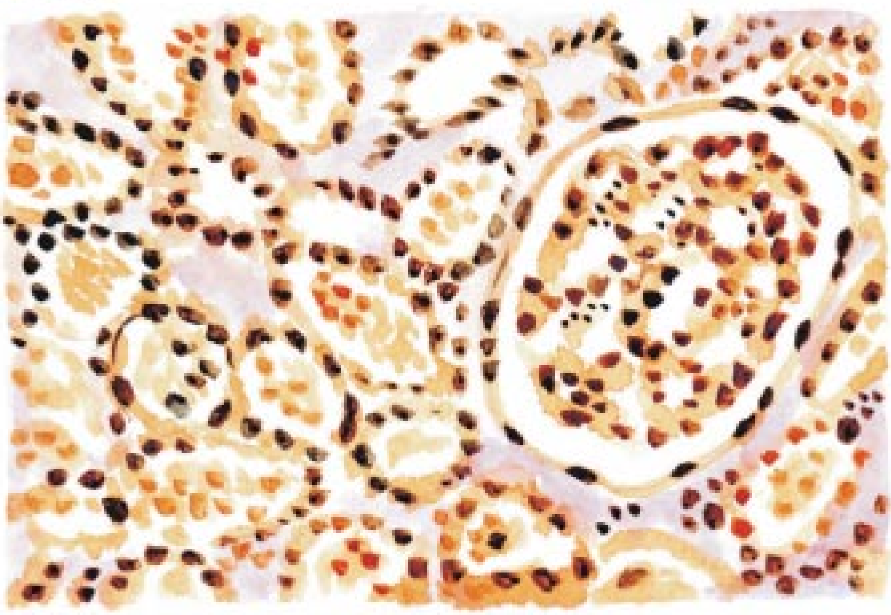
	Prerenal	Kidney type
Urinary density	> 1015	< 1010
Urinary osmolarity (Osm.) Sodium	> 400 mOsm/L	< 300 mOsm/L
concentration in urine Osm. urinary/	< 20 mEq/L	> 20 mEq/L
Osm. plasma urinary urea/plasma	> 1.3	< 1.3
urea	>3	<3





Circulatory and other selected functional disorders in the course of acute renal failure; Urinary flow, clearances, and PAH elimination are much more affected than renal blood flow and oxygen consumption.

Acute kidney failure
parenchymatous: large, pale kidney
With pale and thickened bark
and hyperemic pyramids



Biopsy section: normal glomerulus;
dilated distal tubules with wavy epithelium and containing blood casts

Fig. 67.1 Acute renal failure.

Different theories have tried to explain the pathophysiological changes that occur in chronic kidney failure:

Theory of intact nephrons.

-Theory of the pathogenic mechanisms of uremic manifestations.

Theory of intact nephrons

For many years, the so-called intact nephron hypothesis has predominated, according to which renal failure is a consequence of a marked decrease in the number of functioning nephrons in both kidneys. In advanced stages, only some nephrons remain healthy or intact, which are responsible for eliminating the osmotic load corresponding to the entire nephron population of the healthy subject.

As the population of undamaged nephrons decreases, more nitrogen products are retained and the osmotic load of the remaining nephrons increases. An osmotic diuresis is thus produced, giving rise to isotonic polyuria with the plasma. The kidney does not concentrate or dilute urine, a condition known as isosthenuria.

This stage is usually preceded by the so-called hyposthenuric phase, characterized by hypotonic polyuria, expression of medullary and tubulodistal compromise, with loss of the ability to concentrate urine and preservation of the dilution capacity.

Theory of pathogenic mechanisms of uremic manifestations

Initially, it was believed that hyperazoemia, and within it urea, was responsible for the symptomatic constellation of uremia; For this reason, the term uremia was used as a synonym for renal failure, which is maintained due to the ease of dosing urea and its elevation in renal failure. This deep-rooted conception was challenged with the advent of dialytic methods of extrarenal purification, which showed that the majority of uremic symptoms disappeared, even if a high blood concentration of urea was artificially maintained (by adding urea to the hemodialysis bath).

However, the measurement of plasma urea remains the best marker of uremic toxicity, since patients on chronic dialysis with urea levels greater than 160 mg/dL have a greater number of complications.

With little success, attempts have been made to implicate other nitrogenous compounds present in abnormally high concentrations in these patients, such as phenols, guanidine and certain polypeptides.

There are symptoms dependent on high blood pressure, hydroelectrolyte alterations and the balance. acid-base balance, and symptoms of uremic origin, none of which can be attributed with certainty to the humoral disturbances found in these patients. They are then attributed to a multifactorial mechanism involving dialyzable substances, still unknown, that explain the clinical improvement achieved with artificial purification methods.

It is worth remembering that protein catabolism originates nitrogenous bodies that increase in case of kidney failure, which can be classified into two groups: nitrogen bound to protein; remaining nitrogen or non-protein nitrogen (includes urea, creatinine, uric acid, amino acids and aromatic bodies). Urea represents 50% of this second group, but is not toxic;

Methylguanidine and guanidinsuccinic acid are the most important compounds due to their toxicity. Hyperazotemia is not directly related to filtration alteration. More exact is the relationship that exists between creatininemia, creatinine clearance and glomerular filtration. The rise in creatinine anticipates that of urea, when glomerular filtration is altered. Schematically it is accepted that:

With glomerular filtration less than 60%...
creatinine rises.
With glomerular filtration less than 40%.
urea rises.
With glomerular filtration less than 25%...
the clinical syndrome of renal failure or uremia appears.

SYNDROMOGRAPHY OR POSITIVE DIAGNOSIS

Clinical picture

Background

Previous personal history of nephropathy (hematuria, proteinuria, renal colic, expulsion of stones, urinary infection, edema, arterial hypertension), coma, pregnancy toxemia, excessive consumption of analgesics (containing phenacetin); family history of kidney disease.

Symptoms

We consider useful the scheme that establishes three stages in chronic renal failure:

Compensated phase.
Initial decompensated phase.
- Late decompensated phase: uremia.

Conpensated phase

By definition it is asymptomatic. Only detectable by functional tests (glomerular filtration).

Initial decompensated phase

It is characterized by anemia and arterial hypertension (mild to moderate), hypotonic polyuria and nocturia.

Late decompensated phase: uremia

End-stage chronic renal failure gives rise to a rich symptomatic constellation that includes all organic systems and constitutes a true syndrome:
uremia.

The clinical manifestations of uremia occur when the glomerular filtration rate falls below 25%, if it is slow to occur, since if the drop appears suddenly we will have to look for triggering factors such as: infections, obstructive uropathies, heart failure, accelerated arterial hypertension or hydromineral disorders.

The clinical constellation is expressed by the following manifestations (fig. 67.2):

Skin and subcutaneous cellular tissue manifestations

The cutaneous-mucous pallor resulting from a commonly severe anemia is constant. It may exhibit a certain melanic tinge.

Pruritus is a common and annoying symptom. It is attributed to deposits of calcium salts in the skin, related to secondary hyperparathyroidism that generally occurs in advanced renal failure.

Uremic frost also appears in advanced stages. It consists of small whitish urea crystals that appear on the face, neck and chest.

Renal type edema is common, which can be determined by massive proteinuria (nephrotic syndrome), by a marked reduction in renal sodium and water elimination, or by congestive heart failure.

Respiratory manifestations

Respiratory arrhythmias, such as Kussmaul and Cheyne-Stokes, may occur in relation to metabolic acidosis or heart failure, and dyspnea and wet rales may occur, indicating the presence of pulmonary edema (called uremic lung).

Cardiovascular manifestations

There is arterial hypertension due to sodium and water retention. Sometimes the renin-angiotensin-aldosterone mechanism also operates.

Symptoms of congestive heart failure secondary to hypertension occur.

Pericarditis. The pericardial rub, indicative of the existence of uremic pericarditis, constitutes a serious sign. Pericarditis usually occurs without effusion, its origin is uncertain.

Digestive manifestations

Urinary or ammoniacal breath is characteristic of terminal states.

Anorexia, nausea and vomiting are common, sometimes associated with diarrhea.

Digestive bleeding (hematemesis and nemelena) may also occur due to intercurrent digestive ulcerations.

Hiccups are frequent.

Parotid swelling (uremic parotitis) and oral swelling (uremic stomatitis) are not rare.

Hematological manifestations

Anemia is a must. It occurs due to a deficiency of erythropoietin associated with a shortening of the half-life of the red blood cell (relative erythropoiesis insufficiency syndrome).

Splenomegaly is found in some etiologic forms, such as systemic lupus erythematosus and amyloidosis.

Hemorrhagic diathesis may appear related to a deficiency of coagulation factors and decreased capillary resistance.

Leukocyte dysfunction.

Neurological manifestations

There is insomnia and psychological disorders. In very advanced stages, drowsiness, mental confusion and obtundation occur, which can lead to coma, characterized by restlessness, punctuated by screams and startles.

Physical examination reveals tendon subjumps and asterixis. Babinski's sign is not rare.

Convulsions may appear, determined by water intoxication, hypertensive encephalopathy and dialytic imbalance (osmotic cerebral edema in hemodialyzed patients, caused by delay of urea in crossing the blood-brain barrier).

Nocturnal muscle cramps are a common and annoying symptom.

There may be peripheral neuropathy.

A polyneuritic syndrome can be found in the lower limbs (arreflexia, sensory and motor deficit, muscle atrophies) especially in patients undergoing repeated dialysis.

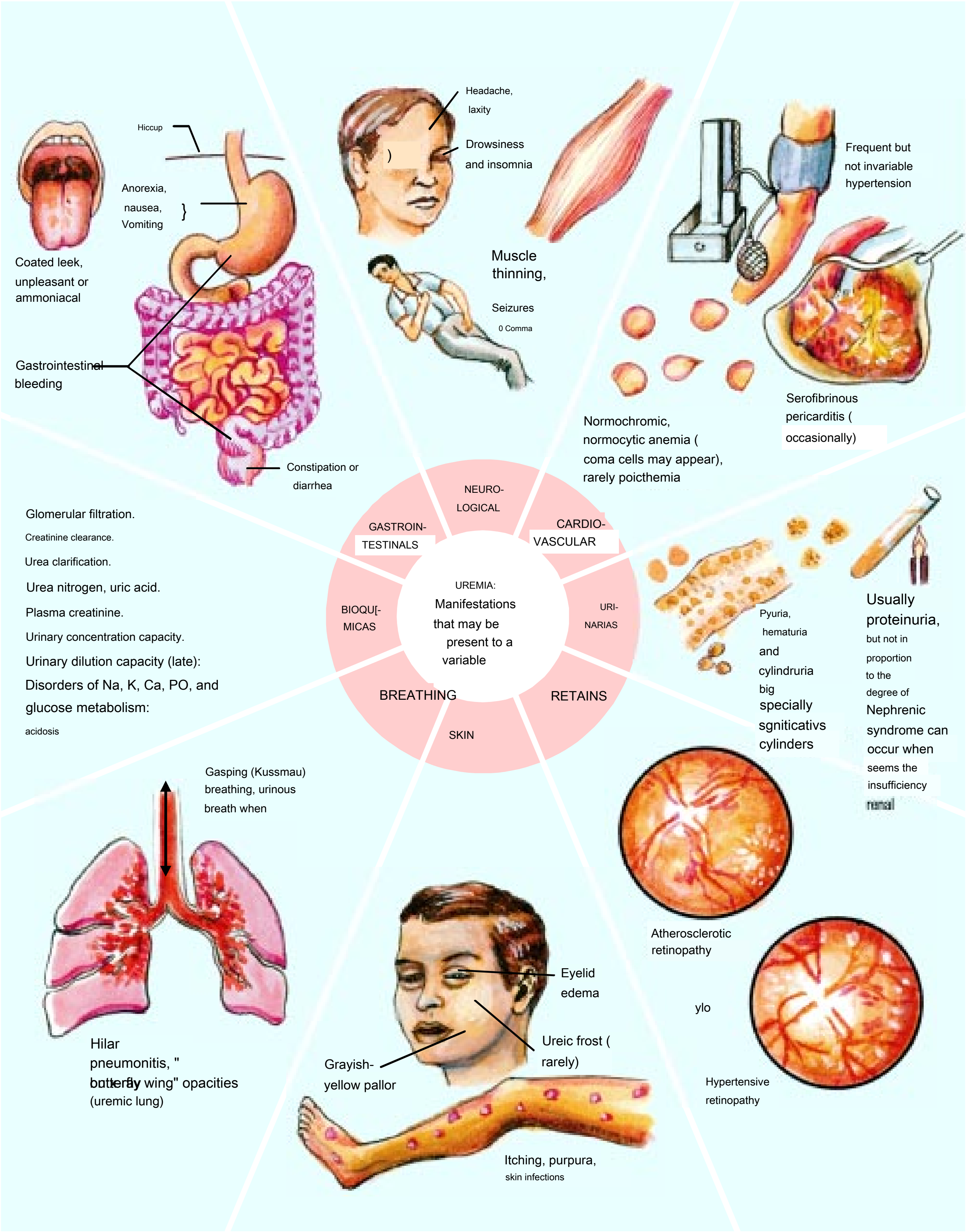

Hiccup
Anorexia,
nausea,
Vomiting
Coated leek,
unpleasant or
ammoniacal
Gastrointestinal
bleeding
Constipation or
diarrhea
Headache,
laxity
Drowsiness
and insomnia
Muscle
thinning,
Seizures
0 Comma
Frequent but
not invariable
hypertension
Serofibrinous
pericarditis (
occasionally)
Normochromic,
normocytic anemia (
coma cells may appear),
rarely poicthemia
NEURO-
LOGICAL
GASTROIN-
TESTINALS
CARDIO-
VASCULAR
UREMIA:
Manifestations
that may be
present to a
variable
BIOQU[-
MICAS
URI-
NARIAS
BREATHING
RETAINS
SKIN
Glomerular filtration.
Creatinine clearance.
Urea clarification.
Urea nitrogen, uric acid.
Plasma creatinine.
Urinary concentration capacity.
Urinary dilution capacity (late):
Disorders of Na, K, Ca, PO, and
glucose metabolism:
acidosis
Pyuria,
hematuria
and
cylindruria
big
specially
sgniticativs
cylinders
Usually
proteinuria,
but not in
proportion
to the
degree of
Nephrenic
syndrome can
occur when
seems the
insufficiency
renal
Gasping (Kussmau)
breathing, urinous
breath when
Hilar
pneumonitis, "
butterfly wing" opacities
(uremic lung)
Eyelid
edema
Ureic frost (
rarely)
Grayish-
yellow pallor
Itching, purpura,
skin infections
Atherosclerotic
retinopathy
ylo
Hypertensive
retinopathy

Fig. 67.2 Clinical manifestations of uremia.

Bone manifestations

Bone pain caused by skeletal injuries may occur in relation to secondary hyperparathyroidism. Pathological fractures, which predominate in the ribs (Milkman syndrome or milk drinkers syndrome), are not rare.

Metabolic and endocrine disorders

1. Renal osteodystrophy.
2. Carbohydrate intolerance.
3. Hyperuricemia.
4. Hypertriglyceridemia.
5. Malnutrition.
6. Decreased growth and development.
7. Infertility and impotence.
8. Amenorrhea.

Muscle disorders

Proximal myopathy may occur.

Complementary exams

Hemogram. Neutrophilic leukocytosis is common, erythrocyte sedimentation rate is high.

Study to detect electrolyte balance disorders:

- Hyperkalemia.
- Hyperphosphatemia.
- Hypocalcemia.
- Metabolic acidosis.

Glomerular filtration (endogenous creatinine clearance) is decreased and urine concentration and dilution tests are altered.

The Addis count is pathological. Proteins in the urine are increased.

ETIOLOGY (FIG. 67.3)

The causes may be:

1. Primary glomerulonephritis.
2. Secondary glomerulonephritis.
3. Diseases of the urinary tract.
4. Interstitial nephritis.
5. Congenital abnormalities.
6. Diabetic nephropathy.
7. Diseases of the renal arteries and high blood pressure.
8. Collagenous diseases: lupus erythematosus, sclerodermia, polyarteritis nodosa.
9. Unknown.
10. Others.

NEPHROTIC SYNDROME

CONCEPT

Nephrotic syndrome is a clinical entity caused by multiple causes and characterized by massive proteinuria, greater than 3.5 g/day, hypoproteinemia with less than 25 g of serine/L of plasma and a variable tendency to edema and hyperlipemia.

SYNDROMOGENESIS 0 PATHOPHYSIOLOGY (FIG. 67.4)

Nephrotic syndrome invariably results from an alteration at the level of the glomerular basement membrane, which presents increased permeability to plasma proteins, which cross it in inverse relation to their molecular size.

This results in intense proteinuria which, depending on its massiveness and duration, determines a decrease in proteins in the blood concentration or hypoproteinemia.

Albumin or serine and gamma globulin, which are the lowest molecular weight, decrease; while those with higher molecular weight, such as alpha 2 globulins and fibrinogen, increase.

The origin of hyperlipidemia (hypercholesterolemia and hypertriglyceridemia) is unclear, although it seems pathogenically related to hyposerinemia.

Edema is a consequence of a chain of events in which hyposerinemia plays the fundamental role (Fig. 67.5).

Hyposerinemia decreases the colloid osmotic power of the plasma, favoring the extravascular diffusion of Na and H,0 with the consequent decrease in plasma volume (underfilling theory). This hypovolemia determines an increase in sodium reabsorption in the proximal tubule (glomerulotubular imbalance) and in the distal tubule (renin-angiotensin-aldosterone mechanism: secondary aldosteronism).

While this underfilling theory may explain edema formation in some cases of nephrotic syndrome, recent clinical and experimental experiences suggest that edema formation in many nephrotic patients results from primary retention of sodium by the kidney (overfilling theory).

The intrarenal mechanism responsible for this phenomenon is not well known.

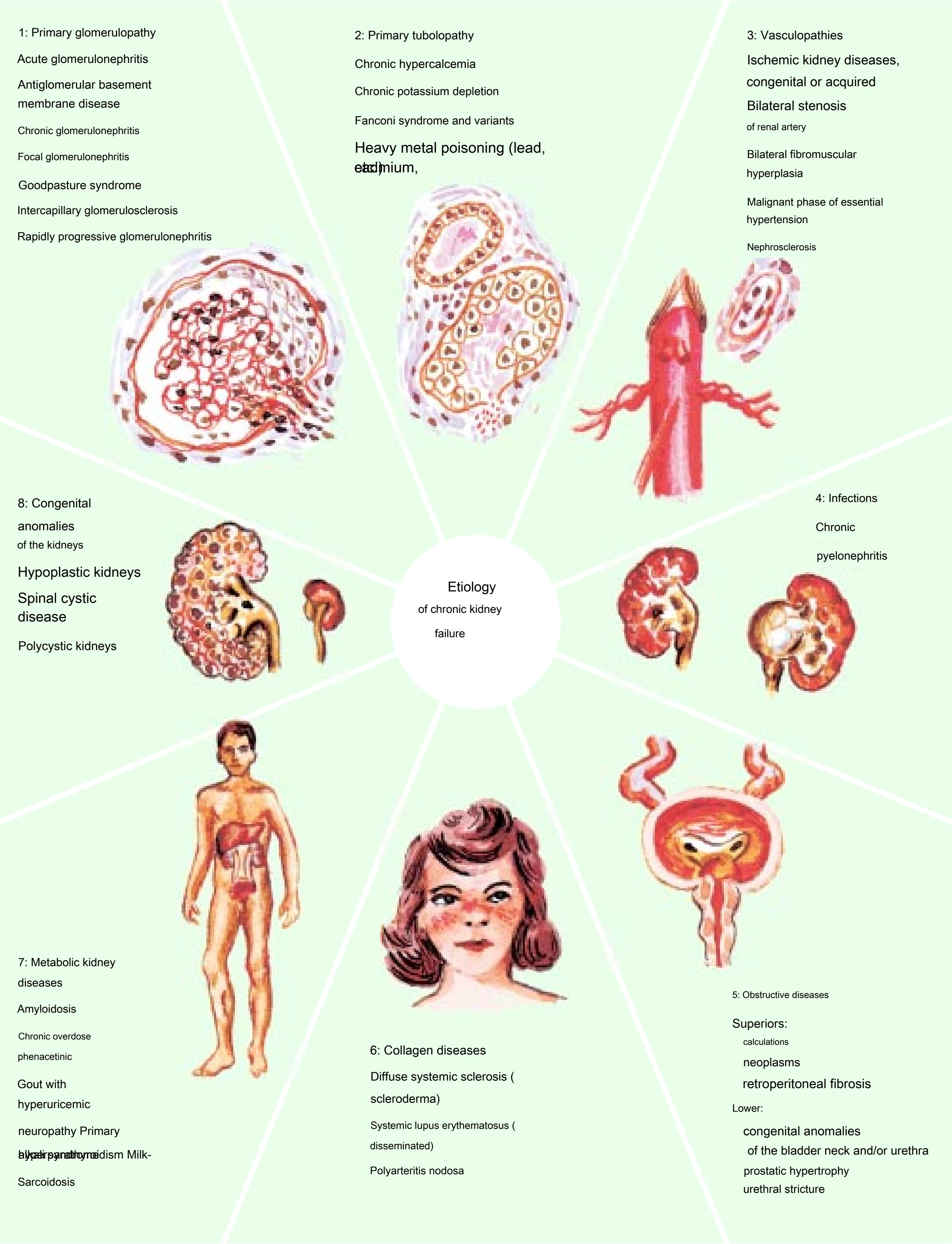

1: Primary glomerulopathy
Acute glomerulonephritis
Antiglomerular basement membrane disease
Chronic glomerulonephritis
Focal glomerulonephritis
Goodpasture syndrome
Intercapillary glomerulosclerosis
Rapidly progressive glomerulonephritis
2: Primary tubolopathy
Chronic hypercalcemia
Chronic potassium depletion
Fanconi syndrome and variants
Heavy metal poisoning (lead,
cadmium, etc)
3: Vasculopathies
Ischemic kidney diseases, congenital or acquired
Bilateral stenosis of renal artery
Bilateral fibromuscular hyperplasia
Malignant phase of essential hypertension
Nephrosclerosis
8: Congenital anomalies of the kidneys
Hypoplastic kidneys
Spinal cystic disease
Polycystic kidneys
Etiology of chronic kidney failure
4: Infections
Chronic pyelonephritis
7: Metabolic kidney diseases
Amyloidosis
Chronic overdose phenacetinic
Gout with hyperuricemic neuropathy Primary
hyperparathyroidism Milk-
alkali syndrome
Sarcoidosis
6: Collagen diseases
Diffuse systemic sclerosis (scleroderma)
Systemic lupus erythematosus (disseminated)
Polyarteritis nodosa
5: Obstructive diseases
Superiors:
calculations
neoplasms
retroperitoneal fibrosis
Lower:
congenital anomalies of the bladder neck and/or urethra
prostatic hypertrophy
urethral stricture

Fig. 67.3 Etiology of uremia.

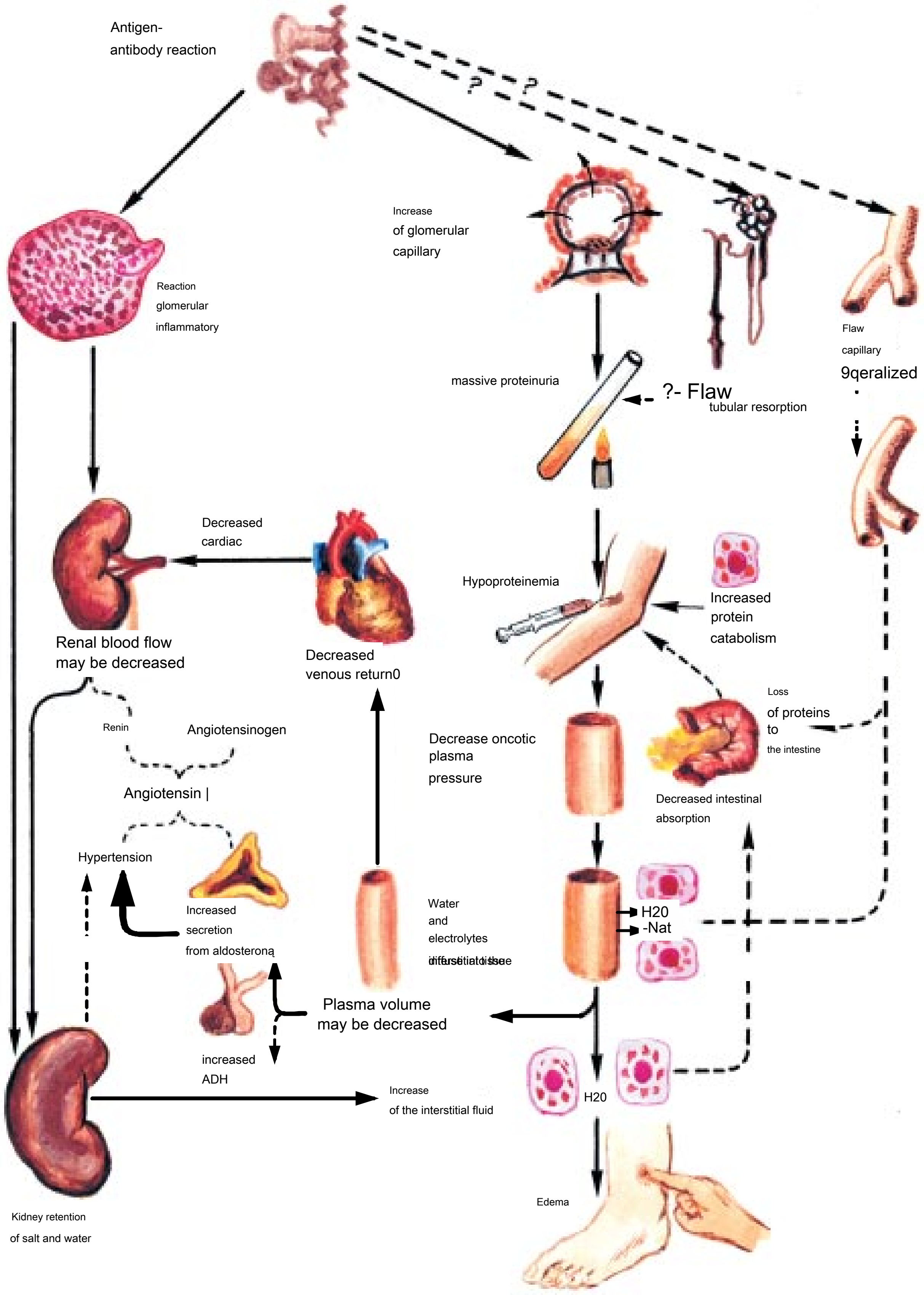

Antigen-
antibody reaction
?
?
Increase
of glomerular
capillary
Reaction
glomerular
inflammatory
Flaw
capillary
9qeralized
massive proteinuria
?- Flaw
tubular resorption
Decreased
cardiac
Hypoproteinemia
Increased
protein
catabolism
Renal blood flow
may be decreased
Decreased
venous return0
Loss
of proteins
to
the intestine
Renin
Angiotensinogen
Decrease oncotic
plasma
pressure
Angiotensin |
Decreased intestinal
absorption
Hypertension
Increased
secretion
from aldosterona
Water
and
electrolytes
diffuse into the
interstitial tissue
H20
-Nat
Plasma volume
may be decreased
increased
ADH
Increase
of the interstitial fluid
H20
Edema
Kidney retention
of salt and water

Fig. 67.4 Pathophysiology of nephrotic syndrome.

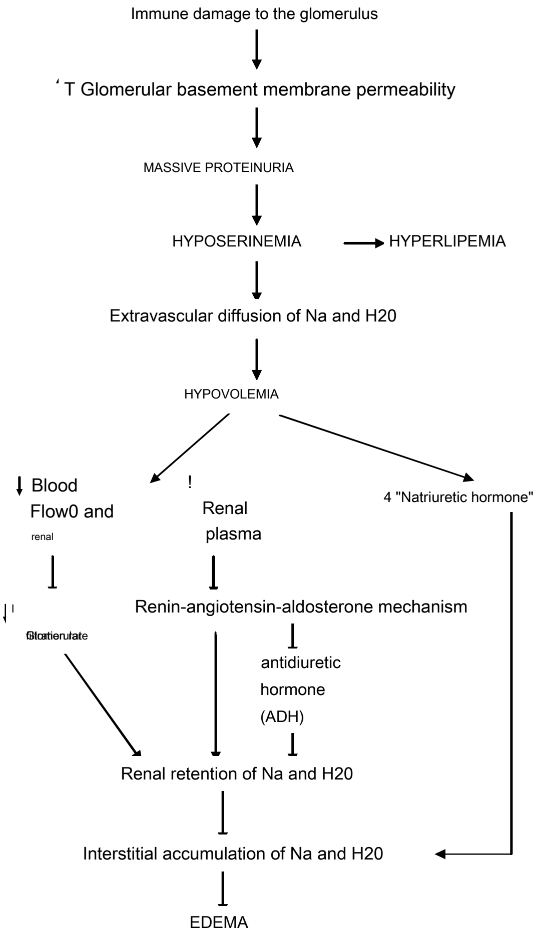

Immune damage to the glomerulus
ʻ T Glomerular basement membrane permeability
MASSIVE PROTEINURIA
HYPOSERINEMIA
HYPERLIPEMIA
Extravascular diffusion of Na and H20
HYPOVOLEMIA
Blood
Flow0 and
renal
!
Renal
plasma
4 "Natriuretic hormone"
Renin-angiotensin-aldosterone mechanism
Glomerulate
antidiuretic
hormone
(ADH)
Renal retention of Na and H20
Interstitial accumulation of Na and H20
EDEMA

Fig. 67.5 Pathogenic scheme of nephrotic syndrome.

SYNDROMOGRAPHY OR POSITIVE DIAGNOSIS (FIG. 67.6)

clinical-humoral picture

In order to provide an integrated and coherent description of the syndrome, we will not segregate its clinical and humoral components.

Proteinuria

It is the decisive manifestation of nephrotic syndrome.

It is present in all cases and is essential to establish the diagnosis. This is profuse, massive proteinuria, in the range of 5 g or more in 24 hours. It is made up, fundamentally, of albumin or serine.

Hypoproteinemia and other plasma protein disorders

Hypoproteinemia occurs at the expense of hyposerinemia. Hyposerinemia, in particular, is also essential for diagnosis. Total proteins are below 6 g% and serine below 3 g%; in most cases, below 2.5 g%.

Electrophoresis of plasma proteins can detect other disorders. In addition to hyposerinemia, an increase in alpha 2 globulins is characteristic, present in practically all cases. Less common is the increase in beta globulins. Gammas are usually normal or low. In the case of hypergammaglobulinemia, the existence of a secondary nephrotic syndrome must be suspected, particularly due to systemic lupus erythematosus or amyloidosis.

Hyperlipidemia

It is related to the increase in low-density lipoproteins (hypercholesterolemia) and very low-density lipoproteins (hypertriglyceridemia). Although it is a characteristic sign, it can be missing, so it is not essential to establish the diagnosis. Some authors consider normocholesterolemic nephrotic syndrome, characteristic of systemic lupus erythematosus.

Edema

Edema constitutes the most striking clinical manifestation of nephrotic syndrome. It represents, however, a secondary pathogenic event, and may be missing in some cases. These are true dry nephrosis, that is, purely humoral nephrotic syndromes.

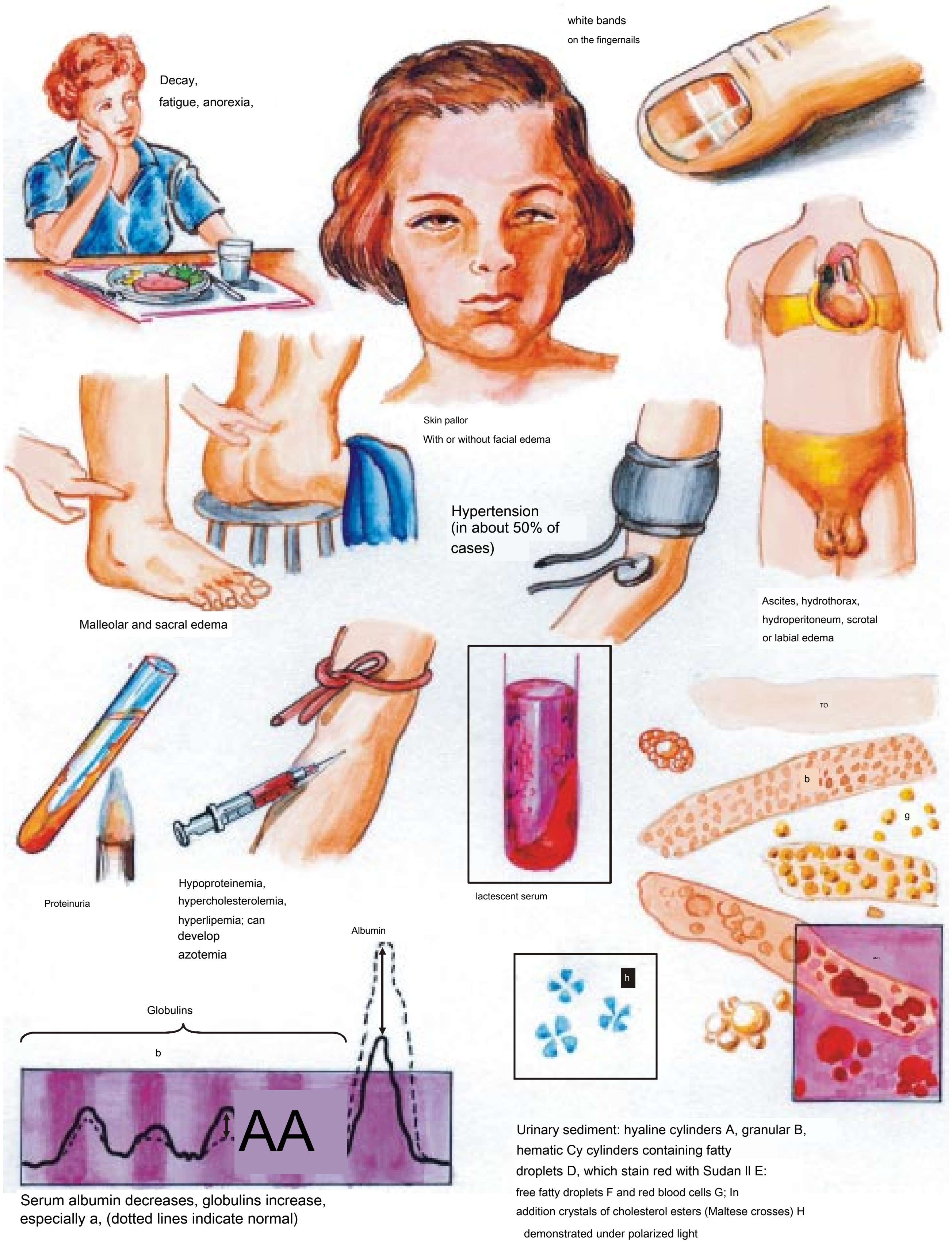
This reservation does not diminish the clinical significance of nephrotic edema, which is characterized by being symmetrical, cold, soft, extremely easy to swallow (butter edema), with almost invariable participation of the face and eyelids (Chinese doll facies) , and other lax areas, such as the penis. and scrotum, and labia majora. It is more marked in the morning. Its intensity is variable, sometimes a true anasarca.

ETIOLOGY (FIG. 67.7)

We can classify nephrotic syndrome as primary, when the kidney is the only damaged organ, and secondary, when the glomerular disease is part of the same hereditary multisystem damage or a primary biochemical error.

Primary glomerular diseases that present with nephrotic syndrome can be of unknown cause or coexist with a well-defined disease, with an infectious, toxic, allergic or tumor cause.

In idiopathic primary nephrotic syndrome, it has been possible to identify the disease with minimal glomerular changes, today better classified as a disease of the epithelial cells of the glomerulus. This entity can be primary or associated with other entities.



white bands
on the fingernails

Decay,
fatigue, anorexia,

Skin pallor
With or without facial edema

Hypertension
(in about 50% of
cases)

Malleolar and sacral edema

Ascites, hydrothorax,
hydroperitoneum, scrotal
or labial edema

Proteinuria

Hypoproteinemia,
hypercholesterolemia,
hyperlipemia; can
develop
azotemia

lactescent serum

Serum albumin decreases, globulins increase,
especially a, (dotted lines indicate normal)

Urinary sediment: hyaline cylinders A, granular B,
hematic Cy cylinders containing fatty
droplets D, which stain red with Sudan II E:
free fatty droplets F and red blood cells G; In
addition crystals of cholesterol esters (Maltese crosses) H
demonstrated under polarized light

Fig. 67.6 Positive diagnosis of nephrotic syndrome.

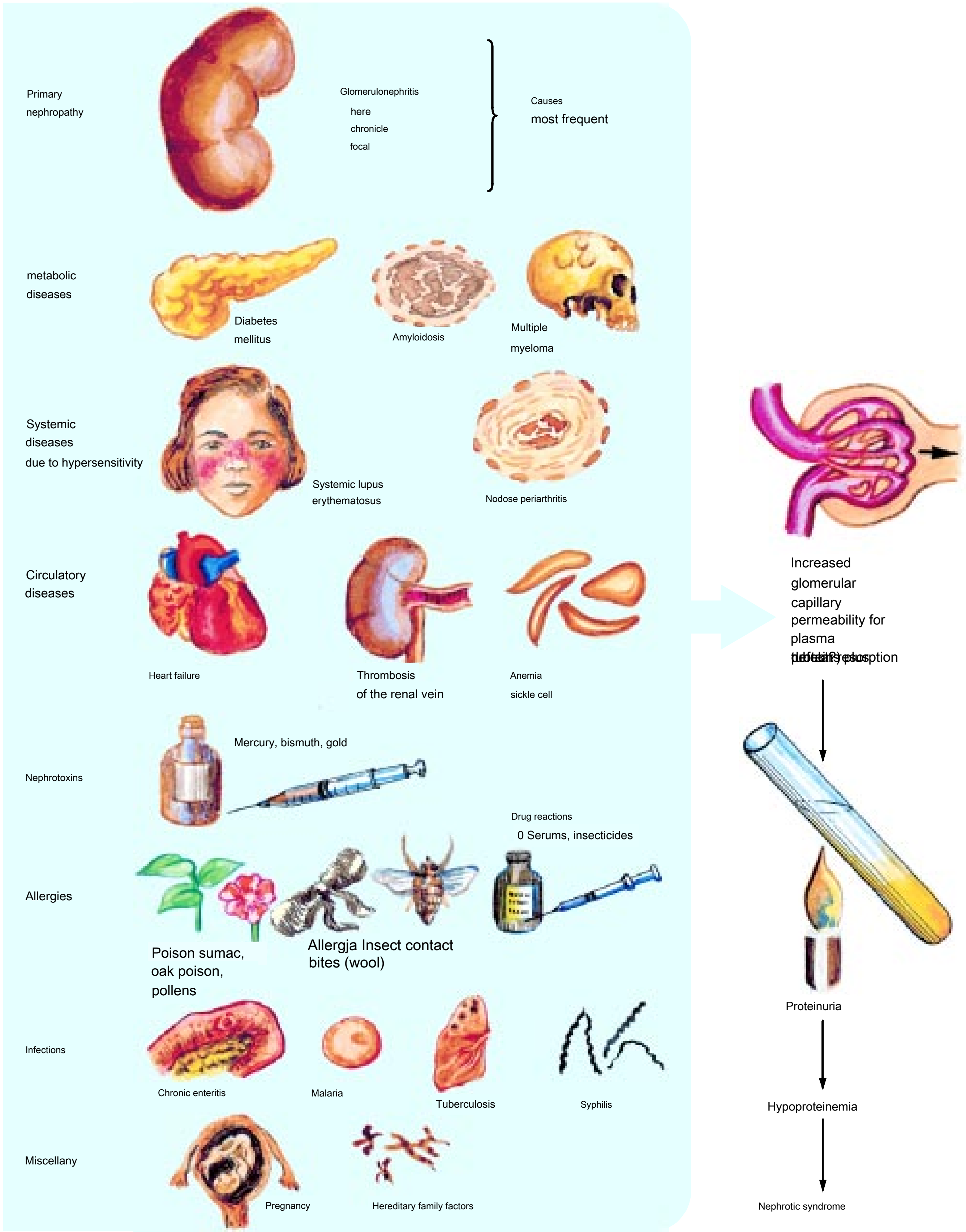

Primary
nephropathy
Glomerulonephritis
here
chronicle
focal
Causes
most frequent
metabolic
diseases
Diabetes
mellitus
Amyloidosis
Multiple
myeloma
Systemic
diseases
due to hypersensitivity
Systemic lupus
erythematosus
Nodose periarthritis
Circulatory
diseases
Heart failure
Thrombosis
of the renal vein
Anemia
sickle cell
Nephrotoxins
Mercury, bismuth, gold
Drug reactions
0 Serums, insecticides
Allergies
Poison sumac,
oak poison,
pollens
Allergja Insect contact
bites (wool)
Infections
Chronic enteritis
Malaria
Tuberculosis
Syphilis
Miscellany
Pregnancy
Hereditary family factors
Increased
glomerular
capillary
permeability for
plasma
proteins (defeat?) tubular reabsorption
Proteinuria
Hypoproteinemia
Nephrotic syndrome

Fig. 67.7 Etiology of nephrotic syndrome.

Primary is known as the complex that makes up three members: the disease with only minimal glomerular changes (lipoid nephrosis), primary segmental and focal sclerosis, and mesangial proliferative glomerulonephritis.

Glomerular epithelial cell disease associated with other entities includes the following:

a) Interstitial nephritis.

b) IgA nephropathy and diabetes mellitus.

c) Nephropathy associated with HIV and heroin.

d) Associated with Hodgkin's disease and other lymphoproliferative diseases.

e) Fabry disease.

68

WATER AND ELECTROLYTES. PRELIMINARY CLARIFICATIONS AND HYDROELECTROLYTIC BALANCE

PRELIMINARY CLARIFICATIONS

CONCEPTS

Weight

The weight of a body is the force with which it is attracted to the center of the Earth.

Mass

It is nothing more than the amount of matter that a body is made of, said in a simplistic way.

Atom

An atom is the smallest amount of an element that can exist in a state of freedom and, at the same time, enjoy its own physical and chemical properties.

atomic structure

The atom consists of a nucleus and a shell. In the first one are the free protons that give it a positive charge and the neutrons (with a neutral charge), which are formed by the union of a proton and an electron. The atomic weight is obtained from the sum of both subatomic particles. In the envelope there are free electrons, with a negative charge, arranged in different layers where they describe different orbits. The number of electrons determines the atomic number on which the chemical properties of an element depend and which has allowed its periodic classification.

In an electrically neutral atom the number of electrons in the shell is equal to the number of free protons in the nucleus.

When an atom gains or loses electrons it becomes an ion, from which it follows that it is nothing more than an electrically charged atom, either positively, if it lost electrons, and in this case it is called a cation, or negatively, if it gained electrons and is now called anion.

Protons and neutrons are united by extraordinarily powerful nuclear forces, which human genius has managed to release; This atomic energy, if used for war purposes, is capable of producing genocides like that of Hiroshima, but if used for pacifist purposes it provides great benefits to humanity.

Proton

Subatomic particle that has been considered the unit of positive charge and has been assigned a weight of 1.

Neutron

Subatomic particle formed by the association of a proton and an electron, with an electric charge equal to zero (0) and a weight also equal to 1.

Electron

Subatomic particle that has been considered as the unit of negative charge and weight 1,826 times less than that of the proton, and therefore, negligible.

There are other subatomic particles such as the neutrino, the positron, etc., which will not be studied in this text.

Atomic weight (p.a)

As already said above, it is nothing more than the sum of the free nuclear protons and neutrons, also present in the nucleus. For example, carbon (C) has 6 free protons in its nucleus and 6 neutrons, therefore, its atomic weight is 12.

Atom-gram

It is nothing more than the atomic weight expressed in grams. For example, the atomic weight of sodium (Na) is 23, therefore its gram-atom is 23 g.

Molecule

It is the smallest amount of a substance, represented by a greater or lesser grouping of atoms, whether of the same nature or of different natures and which can, considered isolated, enjoy those properties that are inherent to it.

Molecular weight (m.p.)

It is nothing more than the sum of the atomic weights of the different elements that make up the molecule in question. Example: sodium chloride (NaCl);
the p.a. of chlorine (Cl) is 35.5 and the p.a. of sodium (Na) is 23; therefore, the p.m. of NaCl is 35.5 + 23 = 58.5.

Molecule-gram (mol)

The mole or gram-molecule is the molecular weight expressed in grams. Example: NaCl; your p.m. is 58.5 and therefore its mole is 58.5 g.

From the concepts of gram-atom and gram-molecule (mol), it follows that both will only coincide when the molecule of the body considered is made up of a single atom, as would happen with the molecules of sodium (Na), calcium (Ca), potassium (K), etc.

Chemical element

An element in chemistry is called those bodies that are indivisible, both by physical and chemical procedures. One could also speak of the so-called elementary substances, which are those formed by atoms of the same nature, and of compound substances, in which the atoms that compose them are different from each other.

The lightest of the known natural elements is hydrogen (H), with an atomic weight of 1, and the heaviest of all is uranium, with an atomic weight of 238.07. It must be clarified that after uranium, which occupies 92nd place in the periodic classification of elements, others have been discovered, logically called transuranide elements.

Isotope

From the Greek isos: equal; topos: place. Two elements are said to be isotopes when, having the same atomic number, they differ in weight, unlike isobaric elements (isos: equal. baros: weight) which, having the same atomic weight, differ in number. It is deduced from the concept of isotopes that what causes the atomic weight to vary is an increase or decrease in the number of neutrons in the nucleus. Example, uranium isotopes: $U^{2}33$, U235 U238 and U239

Valencia

Atomic valence is nothing more than the combining power of a given atom, with respect to others and that in its ionic form, which is the one of interest, is given by the number of electrons that can be transferred or captured at the level of the outermost orbit of the atomic envelope. In the case of giving up electrons, we speak of positive valence, as happens with most metals. If the opposite happens, we speak of negative valence, as occurs with most metalloids or non-metals.

UNITS OF MEASUREMENT

There are three properties of body fluids that can vary in clinical disorders of fluid balance:

1. Volume.
2. Electrolyte composition.
3. Electrolyte concentration.

It is necessary to have certain appropriate units of measurement, either for the volume of liquids, or for the chemical combining power or the osmotic pressure, so that the variations in these properties can be expressed quantitatively.

Volume units

The basic unit of volume of the International System of Units is the liter (L). The cubic centimeter (cm° or cc) has been used for a long time as a measure of volume. Since the centimeter is a linear measurement, it is better, for the purposes of greater clarity and uniformity, to use the liter as the unit of measurement for yolumen.

We can use the milliliter, which is one thousandth (1/1, 000) of a liter. The milliliter, for practical purposes, has the same volume as the cubic centimeter.

Its symbol is mL.

When we want to express 100 mL as a measurement, which is one tenth of a liter, we can use the deciliter, whose symbol is dL.

The term "per 100 mL", that is, the amount of substance in 100 mL of solution, has traditionally been expressed with the symbol percent (%), for example, milligrams percent (mg%), but in most countries it is expressed in dL; example, mg/dL.

Chemical Combining Power Unit

We must choose a unit to measure chemical combining power. The logical unit to measure this effect is the milliequivalent. It is abbreviated mEq. For many years electrolytes have been measured in milligrams. The milligram, however, is a measure of weight and has no relation to the chemical combining power, which is what we wish to measure. If we were to compare the power of a locomotive engine with that of an airplane we would not do so on the basis of weight;

We would better use the horsepower (HP), a unit of power. A locomotive, for example, weighs 100 tons (t). but it only has 1, 500 HP. An airplane, however, weighs only 20 tons, but has 4, 800 HP.

Evidently it is the horsepower, not the weight, that expresses the power of these two types of transport. It is therefore the chemical combining power and not the weight that is important, when we compare electrolytes. Chemical combining power is the same as electrical balancing power, since chemistry and electricity are closely linked in the body' s fluids. It can be said that the milliequivalent represents the horsepower of any electrolyte. The milliequivalent (mEq) is one thousandth of an equivalent (Eq). The latter is defined as the chemical combining power of the weight of a substance, which is equivalent to the chemical combining power of one gram of hydrogen; Therefore, nmiliequivalent refers to the chemical combining power of that weight of an element or ion, which combines or is equivalent to the chemical combining power of 1 mg of hydrogen.

Therefore we can say that the unit of power is the horsepower and the chemical combining power of 1 mg of hydrogen represents the unit of the chemical combining power for the milliequivalent.

An interesting example showing the large disparity between binding weight and chemical combining power is evident when sodium and protein are compared. One liter of plasma contains 3,260 mg of the sodium cation. This amount of sodium has a chemical combining power of 142 mEq: on the other hand, one liter of plasma contains 65,000 mg of the proteinate anion. This amount of proteinate has a chemical combining power of only 16 mEq. From the point of view of chemical combining power, sodium can be compared to an airplane and protein to a locomotive.

One milliequivalent of hydrogen has the chemical combining power of one milligram of hydrogen, the same as 1 mEq of sodium, potassium, chloride, or phosphate.

Likewise, 50 mEq of hydrogen has the chemical combining power of 50 mg of hydrogen, the same as 50 nmEq of sodium, potassium, chloride or phosphate. The same number of milliequivalents in cations chemically bonds with the same number of milliequivalents in anions. There is no fixed relationship between the weight of the cations and that of the anions that are chemically equivalent.

We like to mention the example of dancing cited by Statland. If we were giving a dance for teenagers it would be ridiculous to invite 600 kg of girls and 600 kg of boys. We would create an uncomfortable situation, since it is the number of individuals of each sex and not their weight that matters, from the point of view of girls and boys pairing up for a dance.

The example of dancing is even more useful.

Suppose that girls and boys were invited in equal numbers, as individuals and not as couples. Therefore, any boy can dance with any girl, and any girl with any boy. The same thing happens with our cations and anions. Most of the time they exist as ions rather than as compounds, such as potassium chloride or sodium phosphate. In the wild dance of body fluids, any cation can "dance" with any anion. The only requirement from a chemical point of view is that the total of the cations in milliequivalents is equal to the total of the anions in milliequivalents. The composition of the cation column and the anion column is relatively constant in health. They can, and frequently do, deviate from normal in clinical fluid balance disorders.

Why use milliequivalents?

There are many advantages to the use of milliequivalents in clinical fluid balance problems. 100 mEq of cations always react with exactly 100 mEq of anions. Furthermore, there is no constant ratio in the weight of cations that will react with a weight of anions.

For example, the total plasma cations have a value of 154 mEq/L and are exactly equivalent in chemical combining power to the total plasma anions, which also have a value of 154 mEq/L.

Let us now compare the total weight of the cations with the total weight of the anions in the plasma (Table 68.1).

The first is 558 mg/dL, while that of anions is 6,948 mg/dL.

Conversion from milligrams (mg) to milliequivalents (mEq)

Considering that many laboratory reports express the value of electrolytes in milligrams per 100 mL (mg%, or mg/dL), it is useful to be able to convert such weights to milliequivalents per liter (mEq/L). This can be easily done by employing the following formula:

$$\text{mEq/L}= \frac{\text{mg/dL} \cdot 10.\ \text{valence}}{\text{atomic weight}}$$

The valence and atomic weight of any element can be found in medical dictionaries or chemistry texts. The reader can quickly convert mg/dL to mEq/L, from a practical point of view, multiplying the amount in milligrams per liter by the conversion factor (table 68.2). In this way you can avoid carrying out the mathematical equation.

Electrolyte concentration unit

When expressing the concentration of the solutions, the weight of the dissolved substances must be taken into account, because there may be substances with the same mass and different weights, and vice versa.

If we take hydrogen, which has an atomic weight of 1, and calcium, with an atomic weight of 40, to achieve a solution in which there are the same number of particles of one and the other, we will have to dissolve 40 g of calcium for each gram of hydrogen.

molar solution

The molar solution is one that contains one mole per liter of solution. Example, Ca solution containing 40 g of Ca per liter. A molar solution should not be confused with a molal solution, since the latter contains one mole per liter of solvent.

Molar solutions all have the same number of dissolved particles or, in other words, the same amount of solute.

A millimole is one thousandth of a mole, and a millimolar solution is one that contains one millimole per liter of solution.

Molar solutions have equal osmotic pressure, a term that will be explained below.

Table 68.1 Normal representation of electrolytes in plasma serum

The electrolytes	Voiced by	
	mg/dL	mEq/L
Cations:		
sodium	326.0	142
Potassium	20.0	5
Calcium	10.0	5
Magnesium	2.4	2
Total	358.4	154
Anions:		
Baking soda	60.5	27
Chlorine	365.7	103
Phosphates	3.4	2
Sulfates	1.6	
organic acids	17.5	5
Proteins	6,500.0	16
Total	6 948.7	154

Table 68.2 Normal electrolyte levels in serum or plasma and data necessary for their conversion from mg/dL to mEq/L

The electrolytes	Calculated How	Valencia	Atomic weight	Conversion Factor	Normal figures is mg/dL	mEq/L
Cations:						
Sodium	na	1	23	0.435	315-338	137-147
Potassium	K	1	39	0.256	16-22	4.0-5.6
Calcium	Tot. of Ca	2	20	0.500	9-11.5	4.5-5.8
	Iones Ca	2	20	0.500	4.25-5.25	2.1-2.6
Magnesium	Mg		12	0.833	1.6-2.9	1.4-2.4
Anions:						
Baking soda	Combination power of CO, Cl					
			22.4	0.446	55-65	25-29
Chlorine		1	35.5	0.282	348-375	98-106
	NaCl		58.5	0.171	573-610	98-106
Phosphorus inorganic	Q	1.8	17.2	0.580	3.0-4.5	1.7-2.6
Sulfate inorganic	Yes	2	16	0.625	0.3-2.0	0.2-1.3
acids organic	Acid lactic				14-28	4-8

Conversion between milligrams percent and millimoles per liter

Sometimes it happens that laboratories report the concentration of a certain substance in mg% (mg/dL) and we need it in millimoles per liter (mmol/L); or conversely, with the adoption of the International System of Units, they are expressed in mmol/L and we need it in mg/dL.

The conversion is easy in both cases, if the molecular weight (m.p.) of the solute is known:

$$\frac{\text{mg/dL } 10}{\text{p. m.}} \quad \text{mmol/L}$$

$$\frac{\text{mmol/L} \cdot \text{p. m.}}{10} \quad \text{mg/dL}$$

Osmotic pressure unit

The membranes of the capillary walls are generally permeable membranes, that is, they allow water, electrolytes and certain small molecules to pass through, but not large ones, for example, proteins. This is precisely why the intravascular fluid (plasma) has ne large amount of protein and yet the interstitial fluid contains practically none. This fact explains the mechanism of reabsorption in the venous sector through the oncotic pressure that the proteins develop.

On the other hand, semipermeable membranes only allow water to pass through and no other colloid or crystalloid substances. However, the cell membrane has special characteristics that make it a biological membrane and not a physical membrane; since it may not allow small ions such as potassium to pass through and yet allow large molecules such as glucose to pass through.

That is why it has been said that the cell membrane is a "facultative" membrane, which allows what it wants and when it wants to pass through.

According to these concepts we have that when a liquid containing a relatively small amount of dissolved particles is placed on one side of a semipermeable membrane and on the other side, a liquid containing a relatively large amount of dissolved particles, a force comes into action that balances the concentration of the dissolved matters in the two liquids. This force is known as osnotic pressure.

The osmotic pressure in action is called osmosis. Osmosis can be considered as the extraction power for water, exerted by a concentrated solution located on one side of a semipermeable membrane, which extracts the water from the diluted solution, located on the other side. This "extract" property is completely dependent on the relative concentrations of non-diffusible particles in each solution. Of course, when Osmosis occurs, the volume of the more concentrated solution will increase as water from the less concentrated solution is absorbed through the semipermeable membrane.

The unit of osmotic pressure is called osmol and the most used subunit is milliosmol.

The osmotic pressure exerted by one mole in solution is called osmol and, consequently, the pressure exerted by one millimole in solution is called milliosmol.

Osmolality and osmolarity

The osmotic pressure of a solution (given by the concentration of its solute) is called osmolality, when it is expressed in milliosmoles per kilogram of water; and it is called osmolarity, when it is expressed per liter of solution.

Since the osmotic pressure of a very concentrated solution is related more to its osmolality than to its osmolarity, although in very dilute solutions of the human body the terms may be interchangeable, it is preferred to use the term osmolality.

Confusion occurs because it is easier to refer to the amount of body fluids in liters of solution than in kilograms of water and because it is often spoken in terms of osmolality, even though the calculations are made and expressed in terms of osmolarity (mosm/L).

WATER BALANCE

There is nothing more natural than prescribing saline solution to a dehydrated subject or restricting drinks to an edematous patient. However, behind this apparent simplicity, fearsome difficulties are hidden. Water balance disorders are actually extremely complex.

That is why this chapter begins with a brief summary of the basic aspects of the topic.

Water is the most important element for life, along with oxygen. Man can live several weeks without food, but a few days without water.

It is striking that the amount of this universal solvent is always the same in the body, as is that of the electrolytes.

Life appeared in the sea, which had adequate conditions for it: little variation in its temperature, pH, viscosity, etc., despite the enormous existing atmospheric variations.

Man's extracellular water offers great similarities with the water of primitive seas, which was one third less saline than that of today's seas (this poor salinity increased over the course of millions of years, with the minerals from the rivers).

To maintain the fixed proportion of water and salts in terrestrial animals, complex neurohormonal and hemodynamic mechanisms have had to be perfected.

monkeys

Man needs to drink water from the outside, since the amount he receives by virtue of the chemical reactions that occur in his body is completely insufficient to satisfy his needs and, in addition, he must compensate for the losses by perfecting the excretory mechanisms, of which the most important is the function of the kidney.

The set of mechanisms that regulate the uniformity of the internal environment offers a high degree of specialization; In addition to the kidney, the lungs, the endocrine system and the buffer systems intervene in this regulation.

REGULATION OF WATER AND SALTS

In an adult, water makes up 60-70% of body weight.

The water and the substances dissolved in it make a continuous journey through three compartments - intracellular, interstitial and vascular - bringing the oxygen and nutritional substances it needs to the cell and carrying away waste products (fig. 68.1). The fluid contained in these three compartments are called intracellular fluid, interstitial fluid and blood plasma, respectively.

Approximately one third of the total body water is extracellular and the remaining two parts are intracellular.

The child has a greater proportion of water, it constitutes up to 75% of body weight in newborns (fig. 68.2).

The total amount of water is influenced by the total amount of fat, since the latter hardly has any water, and therefore the more fat, the less water; This means that the proportion of water in women is lower than in men (see fig. 68.2).

The extracellular fluid is divided into two components: the interstitial fluid and the circulating blood plasma. Plasma and the cellular elements of blood - mainly red blood cells, which together constitute the total blood volume - fill the vascular system.

Interstitial fluid is the portion of extracellular fluid that bathes the cells and remains outside the vessels. Transcellular fluid must be added, which includes cerebrospinal fluid, gastric secretions,

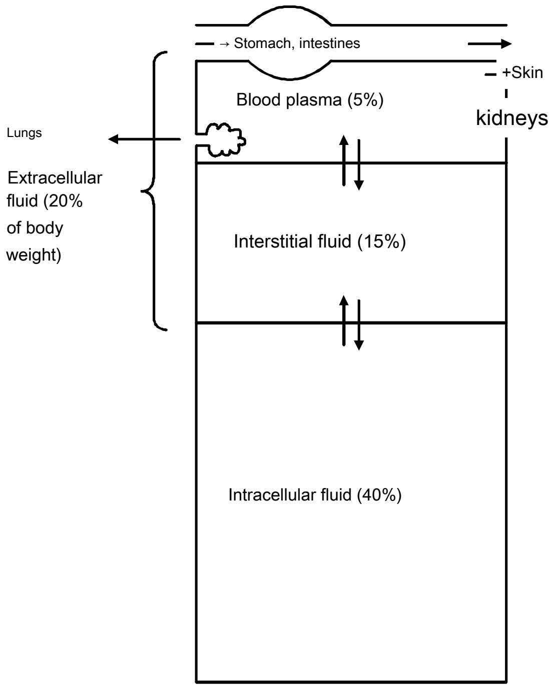


Fig. 68.1 Distribution of water in the body compartments.

hepatic and intestinal fluids, ocular fluids and fluids from potential spaces, such as those in the serous cavities.

The cell lives in an aqueous medium, and itself is largely made up of water.

There is constant entry and exit of fluids that are exchanged between the cells and the interstitial fluid. The conservation of the intracellular and interstitial compartments is achieved mainly, indirectly, by mechanisms that depend on blood volume and its osmolality.

The fluid moves freely between the interstitial and vascular compartments and each buffers the losses or excesses of the other. This change depends mainly on the hydrostatic pressure within the microcirculation and the osmotic tensions of the blood and interstitial fluid.

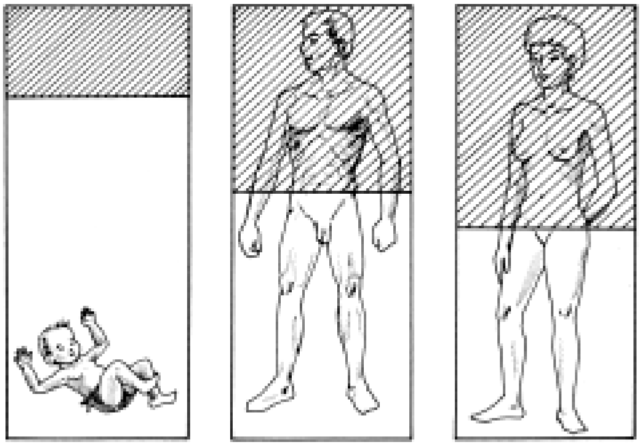


Fig. 68.2 Proportion of the body's total water according to age and sex.

Infant 70-83%

Male 53-60%

Women 45-50%

The volumes and distribution of body fluids are homeostatic constants finely preserved by gains or inputs and losses or outputs.

Gains or income from body fluids

Abnormal differences between the entry and exit of body fluids are the causes of clinical disorders of hydromineral balance.

Liquids composed of water and electrolytes in various proportions are acquired in various ways, which are expressed in table 68.1.

Table 68.1

Ways to gain water and electrolytes
Water and liquids ingested Food
Tube feeding Parenteral feeding Rectal feeding

The water intake is approximately 2.5 L per day, distributed as follows:

1. Visible water (water itself and liquid foods): 1,200 mL.
2. Water from solid foods: 1,000 mL.
3. Water not ingested, metabolic or oxidation: 300 mL.

Obtaining pure, uningested water is possible by:

Food oxidation.

Oxidation of body tissues.

Losses or discharges of body fluids

The list of the various ways in which fluids are lost is much longer than that of gains.

Losses are equal to income; Therefore, normally, they are also around 2.5 L in 24 hours, distributed as follows:

1. Insensible or imperceptible respiration: 1,000 mL, of which about 400 mL are lost through respiration and 600 mL through the skin.
2. Feces: 100 mL.
3. Urine: 1,400 mL.

To insensible perspiration we must add sensitive perspiration, represented by sweating. Sweat is a hypotonic solution of Nay K, while insensible perspiration water without salts is lost.

So water alone is lost exclusively by:

- The skin: insensitive perspiration.
- The respiratory system: in the water vapor of the spine ration.

The state of fluid balance in a healthy adult can be easily determined by comparing the volume ingested as liquid and the volume of urine.

They are usually almost the same.

This simple rule has value, because the other main sources of fluid supplies in health (water extracted from solid food and water obtained by chemical oxidation in the body) are approximately equivalent to the normal loss of water through the lungs and skin plus the water contained in defecation.

As expected, this rule cannot be applied to sick people (children, adolescents and adults).

The water and electrolytes that can be lost in an individual (healthy or sick) are shown in Table 68.3.

In illnesses, when the intake of liquids and solid foods is suspended or reduced, income is exceeded by losses. The patient can then easily lose half a liter more per day. Iary losses are cumulative and a serious deficit in fluid volume can occur relatively quickly, even without abnormal elimination of body fluids such as those in cases of diarrhea and vomiting. When these abnormal losses occur, the patient may be in danger of dying in a matter of days or even hours.

The loss of fluid from the body through other routes such as fistulas, intestinal aspiration, vomiting, diarrhea or profuse sweating, not only causes the loss of water, but also of variable amounts of electrolytes, which It depends on the composition and quantity of the liquid that is removed. The body's secretions and excretions vary widely, both in total electrolyte concentration and electrolyte composition. Therefore, the volume and electrolyte composition of a secretion or excretion must be taken into account. The comparison, in milliequivalents, of the electrolyte composition of the most important secretions and excretions of the body with plasma can be seen in table 68.4.

Summary

1. Abnormal differences between the gain and loss of body fluids are responsible for most of the fluid balance disorders in the clinic.
2. The ways by which water and electrolytes are lost are more numerous than those by which they are obtained.
3. The state of fluid balance in a healthy person can be appreciated by comparing the volume of liquid ingested and the volume of urine, but this rule is not applicable to sick people, since in illness, when food or liquid intake ceases or decreases, losses exceed income.
4. Fluids lost from the body vary widely in their electrolyte composition; Therefore, the volume and source of their loss are important in assessing the patient's clinical status.

Table 68.3 Place and forms of loss of water and electrolytes Place

Form	of loss
Through the eyes:	In the crying
For the skin:	Perspiration, in the exudation of burns, wounds and ulcers due to recumbency and drainage of abscesses.
Through the respiratory tract:	In bronchorrhea and pulmonary edema.
Through the vascular system.	In hemorrhages.
By the gastrointestinal system:	In saliva, vomiting, gastric aspirate, normal defecation, diarrhea, enterostomy, caecostomy and intestinal fistulas.
By the urinary system:	In the urine.
For the mammary glands:	In the milk.
Through the lymphatic system:	In lymphatic duct fistulas.
By the central nervous system:	In the cerebrospinal fluid and spinal or ventricular drains.
Other possible losses:	Thoracic puncture. Abdominal puncture. Internal losses. Formation of edema in traumatized or burned areas. Extensive urticaria.

Table 684 Electrolyte composition of body losses

Losses	MEq mEq mEq			mEq	mEq
most frequent	Na+	c	K	Cat+	HCO,
Urine	75	100	50	15	
feces	20	15	10		
Sweat	60	55	25		
Saliva	33	34	19	3	15
gastric aspiration	75	110	12	3.5	7
Intestinal aspiration Biliary	115	100	10	4	31
fistula	145	90	5	2.5	40
Pancreatic fistula	145	85	5	2.5	120
recent Ileostomy	110	105	20		
Liquid diarrhea	60	60	50		
Cecostomy	60	50	10		

Neurohormonal control of water

The conservation of an adequate hydrosaline balance is important to guarantee the survival of the animal organism. There are numerous regulation systems that guarantee the conservation of water volume and electrolyte concentration; Plasma volume regulation systems (volemia) play a prominent role.

The correction systems are verified based on osmotic and pressor information, and are exerted on both the water volume and the saline concentration, mainly on the sodium concentration, since sodium salts represent approximately 90% of the plasma electrolytes and are the most important osmotically active solutes.

It can be said that the level of plasma sodium is the fundamental factor that determines volume. This is why, when correcting volume, we must consider both water and sodium conservation, for which there are neurohormonal regulations that are related to each other.

The kidney is the main regulator of the total amount of water. If a lot of liquid is ingested, diuresis increases, and if there is dehydration, it decreases. This is achieved by the effect that antidiuretic hormone (ADH) exerts on the distal convoluted tubules and collecting ducts.

This hormone's function is to permeabilize these tubes and thus promote the reabsorption of water.

Normally 180 L of fluid are filtered by the glomeruli every day, while the volume of urine produced is only 1.5 L.

80% of the filtered fluid is necessarily reabsorbed through the proximal convoluted tubule. Water passively leaves this tubule due to osmoic gradients created by the active transport of solutes.

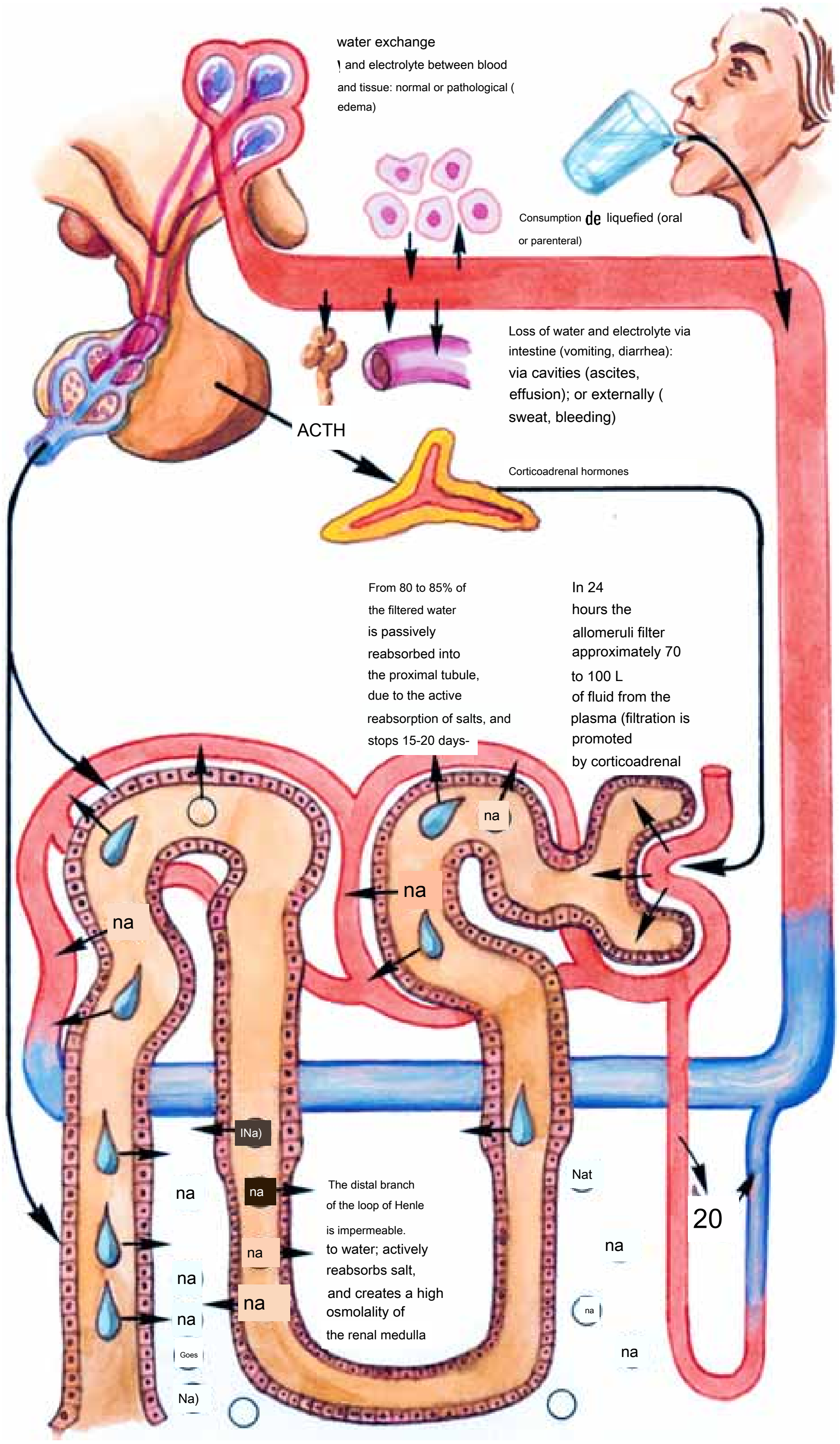
For the rest of the filtered water there is a facultative reabsorption that depends on the action of ADH in the distal tubule and collecting ducts.

ADH is manufactured by neurosecretory cells located in the supraoptic and paraventricular nuclei of the hypothalamus, whose axons do not synapse with nerves or other cellular elements, but end up in the neurohypophysis, ending in bulges aligned around the capillaries. ADH, in the form of secretory granules, uses a carrier protein as a vehicle and migrates along the unmyelinated fibers of the hypothalamic nuclei. It accumulates in the posthypophysis, from where it is released into the bloodstream, promoting the reabsorption of water from the glomerular ultrafiltrate in the distal tubule and collecting ducts (Fig. 68.3).

When ADH is not secreted (as occurs in diabetes insipidus) the volume of water that passes daily into the urine is 5-15 times greater than normal, which makes the extracellular fluids increasingly concentrated. On the other hand, when large amounts of ADH are secreted, water reabsorption increases to the point that the volume of urine formed daily may be only 500 mL, or one-third of normal.

The mechanisms by which ADH is manufactured are of four types:

1. Osmoneurohormonal reflex. The registration of a state of plasma hypertonia by the osmoreceptors located in the supraoptic and paraventricular nuclei determines the release of ADH; The final result of this process is tubular reabsorption of water that tends to correct hypertonia (negative feedback control).



Osmoreceptors in the anterior hypothalamus respond to blood osmolality and regulate the production and release of antidiuretic hormone (

The antidiuretic hormone descends through the nerve fibers and is collected by the capillaries of the neurohypophysis.

Antidiuretic hormone (ADH or vasopressin)

The antidiuretic hormone permeabilizes the distal tubule to water and thus allows its reabsorption along actively reabsorbed salt

Antidiuretic hormone makes the collecting duct permeable to allowing its reabsorption thanks to the high osmolality of the renal medulla

Daily under the influence of antidiuretic hormone they are from 14 to 18 L and is produced 1-2 L of urine in 24 hours

water exchange
\ and electrolyte between blood and tissue: normal or pathological (edema)

Consumption de liquefied (oral or parenteral)

Loss of water and electrolyte via intestine (vomiting, diarrhea): via cavities (ascites, effusion); or externally (sweat, bleeding)

Corticoadrenal hormones

From 80 to 85% of the filtered water is passively reabsorbed into the proximal tubule, due to the active reabsorption of salts, and stops 15-20 days-

In 24 hours the allomeruli filter approximately 70 to 100 L of fluid from the plasma (filtration is promoted by corticoadrenal

The distal branch of the loop of Henle is impermeable. to water; actively reabsorbs salt, and creates a high osmolality of the renal medulla

Fig. 68.3 Metabolism of antidiuretic hormone (ADH).

2. Tensionneurohormonal reflex. Along with plasma hyperosmolality, the most effective physiological stimulus for the release of ADH is the reduction of plasma volume; On the other hand, if the volume of extracellular fluids increases, the production of the hormone is inhibited.

In these cases the osmoreceptors play a secondary role.

Volume variations are recorded by baroreceptors located in the left atrium and great vessels, and are transmitted through the vagus nerve to the supraoptic and paraventricular nuclei that synthesize ADH. The water reabsorption that follows tends to restore blood volume.

3. Osmoneuropsychic reflex. Here the subjective sensation of thirst intervenes, which appears as a consequence of dehydration of the oropharyngeal mucosa.

Intracellular dehydration produced by plasma hyperosmolality determines messages that, coming from the upper part of the digestive tract, can inform the central regulatory structures about the liquid volume ingested. The thirst mechanism is also triggered by information captured by hypothalamic osmoreceptors, which can stimulate the release of ADH.

The hypothalamic structures that intervene in the regulation of thirst are also subject to the influence of other areas of the brain, especially the limbic system.

4. Non-osmotic stinules. In addition to the action of the three reflexes mentioned, other stimuli such as vasodepressor material (VDM), morphine, nicotine and acetylcholine stimulate the production of ADH, while alcohol inhibits it.

ELECTROLYTE BALANCE

Electrolytes or electrolytic substances are those that, when dissolved in water, give rise to the formation of ions (electrically charged atoms), and therefore, are susceptible to decomposition by electric current (electrolysis). Examples: NaCl, HCly H,SO,

Other substances such as glucose and urea do not suffer this ionization phenomenon, and are therefore called non-electrolyte substances.

The distribution of the concentrations of the different electrolytes (cations and anions) in the three body compartments can be seen in figure 68.4.

As we see, the cation that predominates in the plasma is Nat and the anion is Cl. The concentration of the different electrolytes in the plasma and the concentration of the same mos in the interstitial fluid are very similar, except for the protein content, which is much lower in the latter.

The cation that predominates in the cell is potassium (K*) and the anion is phosphate (PO, -).

REGULATION OF THE MAIN ELECTROLYTES

Chlorine and sodium

The normal daily intake of NaCl is 15 g, although 5 g is enough to meet the requirements.

Losses equal income. 90% is lost through urine; the rest, through feces and skin (sweating). If, due to inadequate ingestion or another cause, plasma sodium decreases, it also decreases proportionally in the urine, as occurs in severe hypotonic dehydration; The same thing happens with chlorine.

Most of the sodium is reabsorbed by the proximal tubule (obligate absorption), and the rest by the distal tubule; Adrenal hormones (aldosterone) intervene in the absorption of sodium and chloride by the distal tubule.

It is at this level that the exchange of sodium for potassium and the excretion of the latter occurs.

The factors that control aldosterone secretion (Fig. 68.5) are the following:

1. Stimuli that depend on volume. They arise as a consequence of a decrease in effective arterial perfusion, this is seen in hemorrhages and in states in which plasma volume is reduced. As a result, aldosterone secretion increases via the renin-angiotensin system.

2. Stimuli that do not depend on volume. A group of factors can stimulate aldosterone secretion directly. Angiotensin II can act in the presence or absence of volume deficit. An intimate relationship has been described between potassium balance and aldosterone production. They intervene in addition to the mentioned factors, the action of corticotropin (ACTH) and the decrease in plasma sodium concentration.

3. Physiological levels of ACTH can stimulate aldosterone secretion, but this effect is not as important as the action of potassium and the renin-angiotensin system, which regulate aldosterone secretion through a negative feedback circuit. This phenomenon can be easily understood by looking at Figure 68. 6.

4. In addition to what has been mentioned, the existence of a hormone called adrenoglomerulotropin is assumed, which would be secreted in response to the decrease in cardiac output.

that it would be able to stimulate the production of aldosterone; The pineal gland would be responsible for producing such a

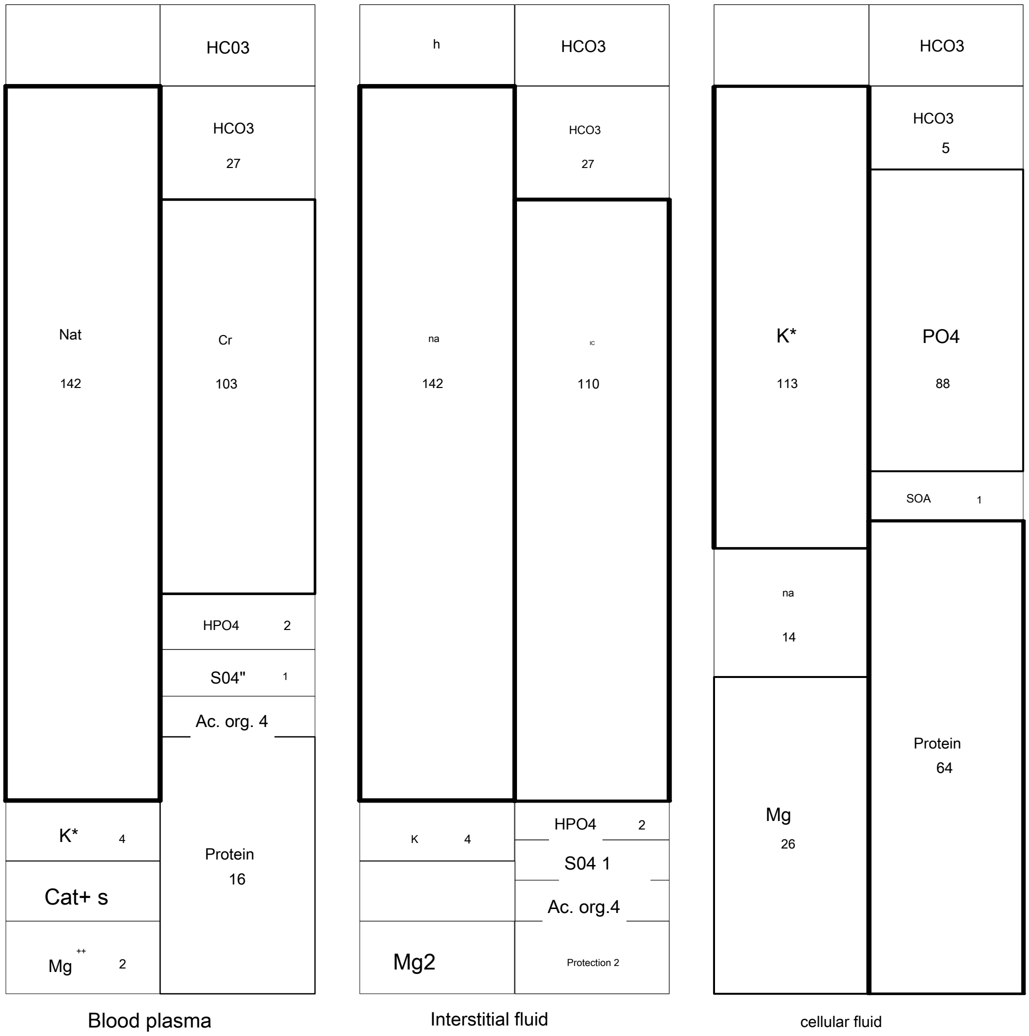
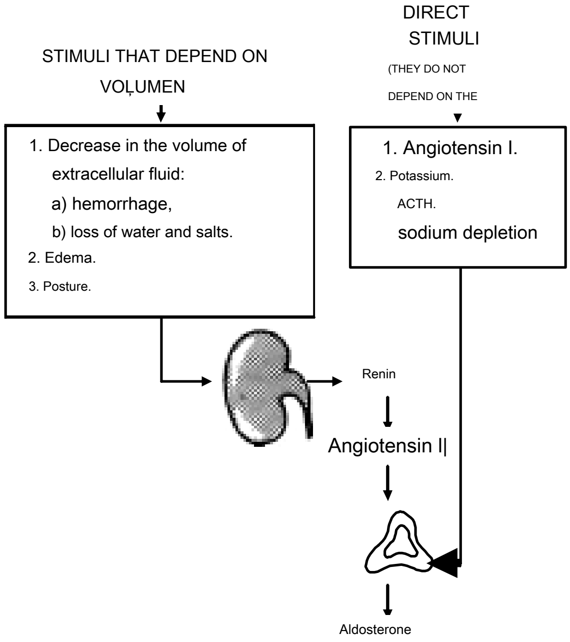


Fig. 68.4 Concentration of electrolytes in the three body compartments, in mEg/ L.

Fig. 68.5 Factors controlling aldosterone secretion.


STIMULI THAT DEPEND ON
VOLUMEN
1. Decrease in the volume of
extracellular fluid:
a) hemorrhage,
b) loss of water and salts.
2. Edema.
3. Posture.
DIRECT
STIMULI
(THEY DO NOT
DEPEND ON THE
1. Angiotensin I.
2. Potassium.
ACTH.
sodium depletion
Renin
Angiotensin I|
Aldosterone

In general, those clinical conditions in which there is a reduction in circulating volume are associated with secondary hyperaldosteronism, even if there is edema, as in nephrotic syndrome.

In the renin-angiotensin system the steps are as follows:

Plasma renin + alphaglobulin → angiotensin |→ angiotensin || → aldosterone

Sodium depletion stimulates the juxtaglomerular apparatus to release renin, while high sodium concentration depresses it. The juxtaglomerular apparatus acts as a volume receptor;

when perfusion or flow is low, it responds by increasing degranulation and renin production; On the contrary, when the pressure is high, its activity decreases.

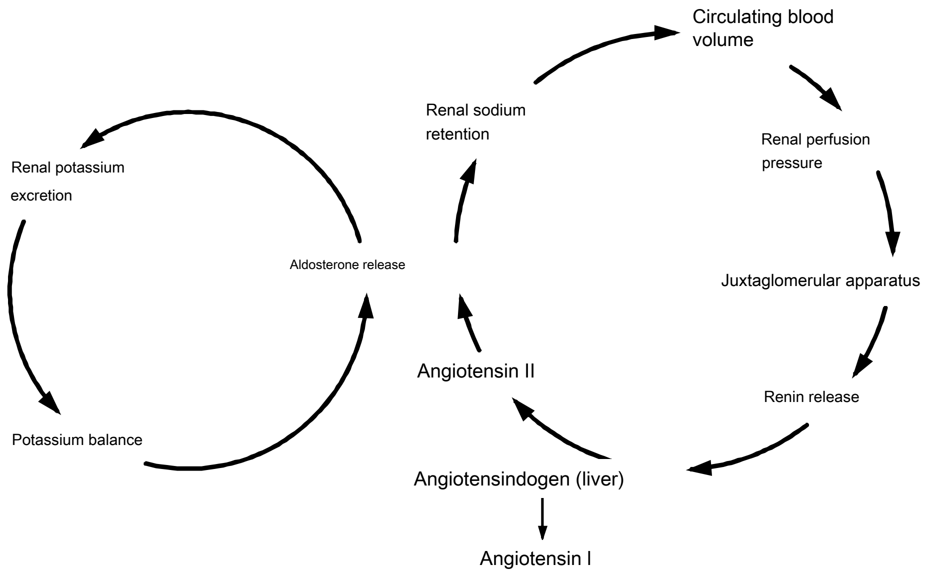


Fig. 68.6 Action of potassium and the renin-angiotensin system on aldosterone secretion.

In Addison's disease, large amounts of sodium can be lost despite hypotension or shock. It is suggested that aldosterone may affect sodium reabsorption in both the proximal and distal tubes.

The high concentration of sodium in the lower parts of the medullary portion of the kidney (loop of Henle, collecting duct and interstitial tissue) explains the countercurrent mechanism. Thanks to the property of the descending branch of the loop of Henle of being permeable to water and sodium, while the ascending branch is only permeable to sodium and not water, the urine that reaches the distal convoluted tube is slightly hypotonic in relation to the plasma. It is at this level where the antidiuretic hormone acts, making it permeable and allowing water to be reabsorbed. Thanks to the countercurrent mechanism, the kidney can reabsorb a certain amount of water with minimal energy expenditure. We know that most water and sodium are actively reabsorbed by the proximal tubule (obligate absorption).

Every time a positive ion is absorbed by the tubules, a state of electronegativity is created in their lumen. Such potential, through the walls of the tubules, immediately causes negative ions, mainly chloride, to diffuse across the membrane to provide electrical neutrality.

The secretion of aldosterone by the adrenals directly increases sodium reabsorption and at the same time, of course, indirectly causes absorption of the accompanying negative ion; Since approximately three-quarters of the negative ions in the glomerular filtrate are chloride ions, aldosterone is often said to cause chloride reabsorption.

In the next chapter we will see the importance of chlorine in the acid-base balance.

Mineralocorticoids also allow the regulation of NaCl through sweat, and acclimatization to heat has been described: at first a lot of NaCl is lost through sweat, but in the long run the body produces sweat that is poor in this salt.

Among the functions of chlorine and sodium, we note that they:

1. They help preserve the volume of the different compartments by providing 80% of the osmolar concentration. Sodium is the main cation of the extracellular fluid, and its concentration is taken as a reference index to diagnose the different types of dehydration: hypertonic, when its figure is above 150 mEq/L, hypotonic, when it is below 130 mEq/L, and isotonic, between 130 and 150 mEq/L.

2. They are part of the composition of digestive juices. Normally, around 8 L of fluid are secreted by the digestive system, which are absorbed (internal circulation of water and salts); This means that there is a constant formation of these elements without them being spent. Pathologically, vomiting can produce an alkalosis due to loss of IC and H, and diarrhea an acidosis, since sodium (Na') is lost and with it, bicarbonate (HC0,).
3. They help regulate the acid-base balance. The ratios between Cl, HCO, and Na are linked to pH; This increases at the beginning of digestion, since Cly H is released into the gastric juice (alkaline tide); Subsequently, Nat and HCO leave the intestine to form alkaline digestive juices (acid tide).

Chlorine penetrates the interior of the red blood cells when the plasma becomes acidic, which constitutes the phenomenon

Hamburger or the so-called chlorine transfer; The opposite occurs in alkalosis. For this reason, certain value has been given to the determinations of globular and plasma chloride in acid-base balance disorders. However, these dosages are not always useful, which is why they have lost a lot of value as a guide for the diagnosis of these alterations. Sodium regulates neuromuscular excitability; This depends on the ionic concentration of sodium, potassium, calcium and magnesium; Na and K* increase neuromuscular irritability, while Ca*, Mgtt and H", decrease it, according to the following formula:

$$\frac{Na^{*}\ K+}{Cat + Mg^{*} * H+} \qquad K\ (constant)$$

Potassium

It is the most important of the intracellular cations and is present in almost all foods, especially vegetables.

Almost everything is eliminated through urine. Potassium filtered by the glomerulus is completely reabsorbed at the proximal tubule; Consequently, the potassium that appears in the final urine is an excretion product of the distal tubule where it is exchanged with sodium by the action of aldosterone; That is why the greater the elimination of sodium by the glomerulus, the more potassium will be lost.

Among the functions of potassium we have that:

1. Helps maintain intracellular osmolality and electroneutrality: in addition, it is necessary for the proper functioning of intracellular enzymes.
2. It intervenes in the conduction of nervous impulses, both in the voluntary and involuntary nervous systems.
3. It is necessary for the proper functioning of skeletal muscles. We already saw the action it has on neuromuscular irritability.
4. It intervenes in intestinal peristalsis; That is why paralytic ileus appears in hypokalemia.
5. It has an anticurare action.
6. It is necessary for the proper functioning of the heart muscle. Its increase can cause cardiac arrest in diastole, since potassium decreases myocardial excitability; The decrease in potassium can stop the heart in systole, due to increased myocardial irritability.
7. It is related to the acid-base balance and the pH of the blood. In cases of acidosis there is hyperkalemia, since the excess hydrogen ions in the extracellular space migrate into the cells and are exchanged for potassium, while in cases of alkalosis there is hypokalemia, due to

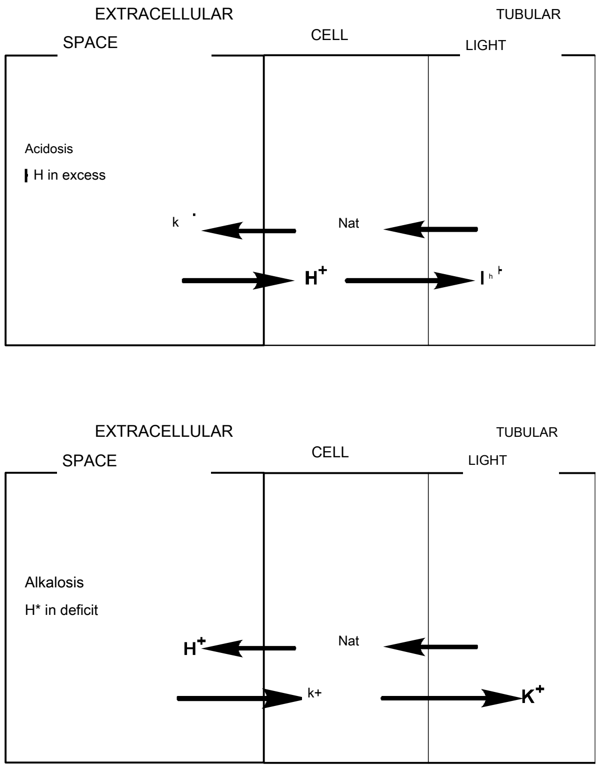


Fig. 68.7 Potassium alterations related to variations in the acid-base balance.

In this case, the hydrogen ions migrate from the cell to the extracellular space, while the potassium from this space passes into the cell and consequently produces hypokalemia (fig. 68.7).

Magnesium

Its concentration in cells is much higher than in the extracellular fluid, indicating its important role in intracellular functions; It is needed as a catalyst for many enzymatic reactions, particularly those related to carbohydrate and protein metabolism.

An increase in the extracellular concentration of Mgt+ depresses the activity of the nervous system and the contraction of skeletal muscle.

A low concentration, on the contrary, increases the irritability of the nervous system and produces peripheral vasodilation and cardiac arrhythmias.

The body's Mg needs are 18 mEq/L per day. This ion is widespread in all foods. 60% is eliminated through feces and 40% through urine.

Calcium

It is found in the body mainly in the form of calcium phosphate in the bones.

If the extracellular calcium ion increases, cardiac activity can be seriously affected until the heart stops in systole. Hypercalcemia can also cause mental depression.

If the calcium ion decreases, spontaneous discharges of nerve fibers occur that can lead to tetany.

Calcium metabolism is closely related to phosphorus metabolism. Calcium is absorbed through the intestine and this absorption is favored by vitamin D. 90% is eliminated through feces and 10% through urine.

Calcium is found in the blood at a concentration of 10 mg, of which 4 mg is in ionized form; ionization is favored by acidosis. If phosphorus goes up, calcium goes down; Therefore, in kidney failure, where there is phosphate retention, hypocalcemia is almost always present.

Calcium decreases neuromuscular excitability, it has the opposite effect to potassium.

Osteoblasts deposit calcium in the skeleton; This action is favored by exercise; alkaline phosphatase is an index of this activity.

Osteoclasts and parathyroid hormone release it; The main action of parathyroidism is to prevent the reabsorption of phosphates by the tubule.

Calcitonin is a hormone secreted by parafollicular cells, located in the interstitial tissue between the follicles of the thyroid gland in humans, probably equivalent to the ultimobranchial glands of lower animals. This hormone inhibits the resorption tion of bone, decreasing the concentration of calcium ion in body fluids; It therefore has an opposite action to paratolmona.

Milk, eggs and cheese are foods rich in calcium.

Phosphorus

Phosphate is the main anion of intracellular fluids. It has the ability to combine reversibly with various enzymatic systems and other compounds necessary for metabolism (functions of ATP, ADP, phosphocreatine, etc.), hence its importance in cellular activity.

Bone contains a very large amount of calcium phosphate.

Phosphate absorption through the digestive tract is very easy as long as it is not combined with Ca to form insoluble compounds.

On the other hand, the increase in the absorption of Ca, thanks to the action of vitamin D, will mean, as a side effect, an increase in the absorption of phosphate.

The kidney controls the concentration of phosphate in the extracellular fluid by modifying its elimination depending on the amount of plasma phosphates.

Parathyroid hormone significantly modifies the elimination of phosphate by the kidneys. In hyperparathyroidism, more phosphate is eliminated and, on the contrary, when the parathyroid hormone produced decreases, phosphate is retained in the extracellular fluids.

ALTERATIONS IN WATER, ELECTROLYTE AND ACID-BASE BALANCES

69

ALTERATIONS IN THE WATER BALANCE

Alterations in water balance are of two types:

1. Default water imbalance: dehydration.
2. Water imbalance due to excess: hyperhydration.

WATER IMBALANCE BY DEFAULT: DEHYDRATION

Previous considerations

When there is a disproportion between the liquids ingested and those eliminated, in which the water balance is negative, dehydration occurs. For many authors this term is incorrect, since when the body loses fluid, the loss is water, but also electrolytes (salts).

The terminology used by different authors to designate the different varieties of dehydration varies greatly, but we prefer to consider three types:

1. Hypertonic dehydration. When proportionally more water is lost than salts.
2. Hypotonic dehydration. When the loss of salts is greater than that of water.
3. Isotonic dehydration. When water and salts are lost in parallel, without altering the relative concentration.

As sodium is the main cation of the extracellular fluid, its concentration is taken as a reference point to know if the amount of dissolved substances is increased (hypertonia or hyperosmolality) or decreased (hypotonia or hyposmolality), as seen in Table 69.1.

The above is not an absolute rule, since it may be the case that a low concentration of sodium detected in the ionogram may indicate a true loss of that cation or, simply, a transfer of ions from the extracellular to the intracellular space. If sodium depletion is present, the patient should be treated with saline solutions, but if hyponatremia is due to internal mobilization of sodium, the above treatment may be dangerous.

Table 691

Types of dehydration, according to sodium (Na)	
Hypertonic dehydration mEq/L	Na above 150
Hypotonic dehydration mEq/L	Na below 130
Isotonic dehydration	Na between 130 and 150 mEq/L

There is no simple test or reaction that allows us to assess the magnitude of a deficit; hence the importance of constant and careful observation of the patient and their response to treatment. These patients frequently go from one type of dehydration to another, and although it is true that the fluids provided can be life-saving, errors in therapy can cause severe secondary disorders.

In the previous chapter we reviewed the electrolyte concentration of plasma, interstitial fluid and intracellular fluid. Let us now look at that of some other organic fluids, such as urine (Table 69.2) and sweat (Table 69.3).

The fluid secreted in the digestive system is 7-10 L (Table 69. 4); Almost all of it is reabsorbed in the ileum and beginning of the colon, therefore, feces contain little water and electrolytes.

Table 622

Normal figures for electrolytes in urine	
Chlorine	130 mEq/L
Sodium	140 mEq/L
Potassium	35 mEq/L

Table 623

Normal figures for electrolytes in sweat	
Chlorine	50 mEq/L
Sodium	50 mEq/L
Potassium	14 mEq/L

Table 694

Liquids secreted by the digestive system	
Saliva	1 500 mL
gastric juice	2 500 mL
Pancreatic juice Bile	50 mL 500 mL 3,
intestinal juice	000 mL

With diarrhea, sodium and potassium are mainly lost, and with vomiting, chlorine and hydrogen ions [H].

Sweat is a hypotonic solution with respect to plasma, although in copious sweating you can lose up to 75 mEq of sodium per day.

For every degree Celsius that body temperature increases, a loss of 150 mL of water is calculated in 24 hours, and for every five breaths per minute that the respiratory rate increases continuously, 100 mL are lost in 24 hours.

Depletion of pure water always occurs due to a decrease in water intake and that caused by losses is much rarer.

When water ingestion is stopped, aqueous reserves decrease due to the continued obligatory losses through the lung and skin (from 600 mL to 1 L every 24 h), and the continued excretion of a minimum volume of urine (500 mL/day). The only source of water supply is oxidation water (300 mL), a totally insufficient amount for the kidney to excrete metabolic waste products. Death would occur within a week of complete water deprivation.

Having made these considerations, we will now describe each of the types of dehydration:

- Hypertonic dehydration syndrome.
- Hypotonic dehydration syndrome.

Mixed dehydration syndrome.

Hypertonic dehydration syndrome

Synonymy

Simple primitive dehydration, loss of pure water, hyperelectrolytaemia, desiccation and water depletion.

Concept

This syndrome is characterized by the loss of water from an organism with little or no accompanying loss of electrolytes.

Syndromogenesis or pathophysiology

We have said that the total amount of fluid in the body is distributed in three compartments: vascular, interstitial and intracellular, which are closely connected to each other, and that increases or losses of fluid in any of them have an impact on the others, since there is a constant flow and reflux of water and electrolytes.

In this aqueous depletion syndrome, water is lost through the kidney and other excretion pathways, and extracellular water decreases considerably, therefore

There is an increase in blood concentration and, as a consequence, an increase in extracellular osmotic pressure, that is, osmolality. This results in water leaving the cells; Consequently, there is a global loss of water in all tissues, both in the intercellular and extracellular spaces.

This phenomenon can be easily understood by looking at Figure 69.1.

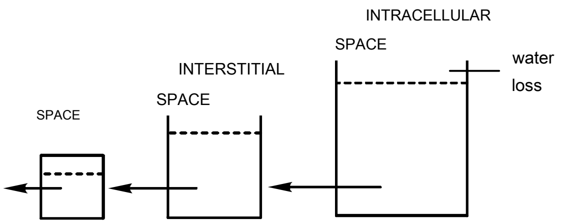

INTRACELLULAR
SPACE
water
loss
INTERSTITIAL
SPACE
SPACE

Fig. 69.1 Hypertonic dehydration.

It is generally accepted, according to Mach, that two-thirds of the water lost is intracellular, not because there is a greater loss of it, but because there is a greater amount of intracellular water compared to extracellular water.

According to Marriott, this syndrome occurs in three stages (fig. 69.2):

In a first stage, consisting of the first and second days without drinking, in which great thirst occurs (cellular dehydration is already evident), a pale grayish facies and a loss of 1-2.5 L of water, condition the loss of 2% of the weight.

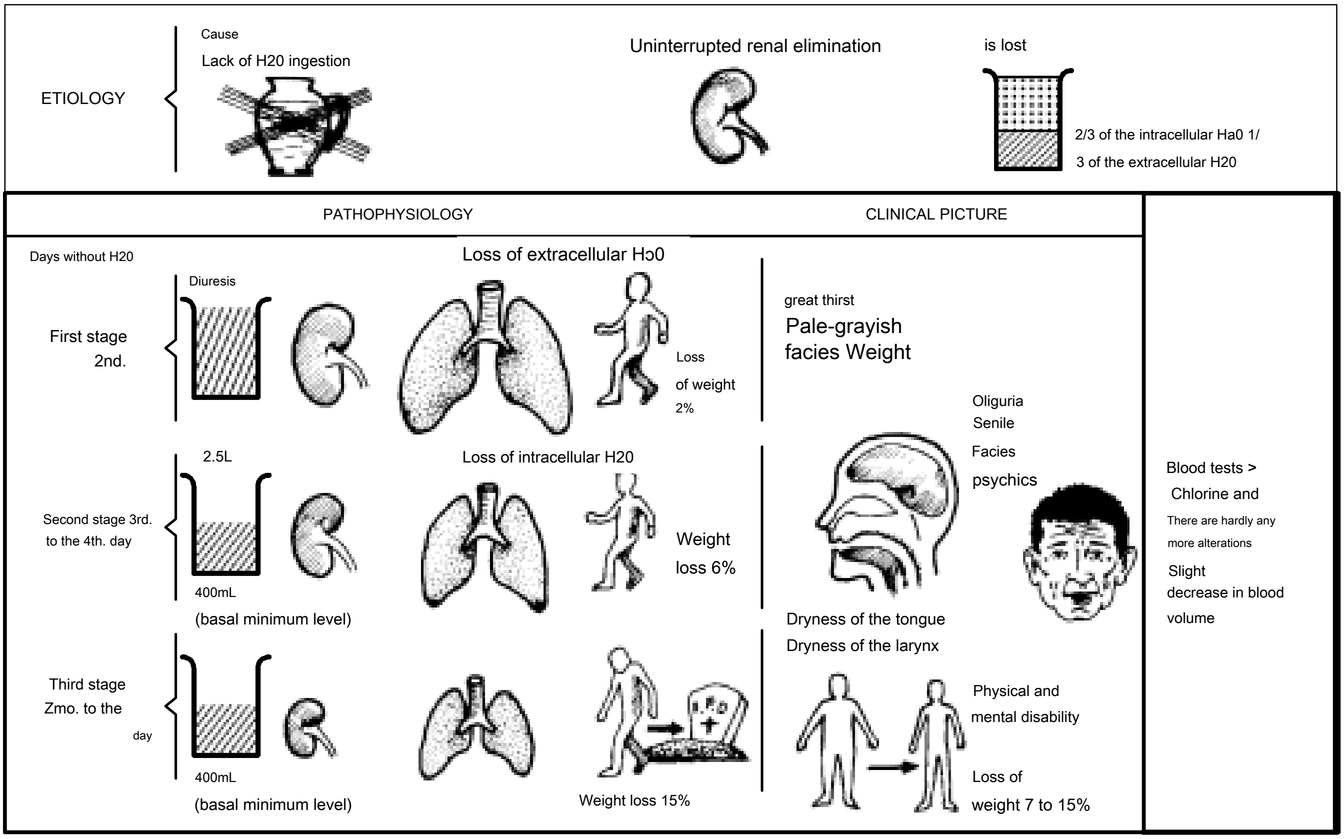
Three or four days after this state (second stage), desiccation increases, causing dryness of the larynx. The timbre of the voice changes, there is manifest oliguria, an appearance of premature aging of the subject and even some psychological disorder is presented.
4L and 6% of body weight have already been lost.

From the fifth day (third stage), a true physical and mental disability of the subject is determined, which enters a state of gravity, with a loss of up to about 10 L of water and approximately 10% of its initial weight. If this state is maintained, death occurs after seven or ten days.

In these cases, the addition of water is very favorable and, on the other hand, the addition of salt is harmful, facts that must be taken into account by the clinician, who must know how to differentiate between these states of water depletion.

In summary, from the pathophysiological point of view, let' s say that in the absence of water intake, extracellular plasma or interstitial water is first lost. This increases the osmotic pressure of the liquid.

Fig. 69.2 Stages in hypertonic dehydration syndrome (loss of pure water).


ETIOLOGY
Cause
Lack of H20 ingestion
Uninterrupted renal elimination
is lost
2/3 of the intracellular Ha0 1/
3 of the extracellular H20
PATHOPHYSIOLOGY
CLINICAL PICTURE
Days without H20
Diuresis
Loss of extracellular Hɔ0
First stage
2nd.
Loss
of weight
2%
great thirst
Pale-grayish
facies Weight
Oliguria
Senile
Facies
psychics
2.5L
Loss of intracellular H20
Second stage 3rd.
to the 4th. day
Weight
loss 6%
400mL
(basal minimum level)
Blood tests >
Chlorine and
There are hardly any
more alterations
Slight
decrease in blood
volume
Dryness of the tongue
Dryness of the larynx
Third stage
Zmo. to the
day
400mL
(basal minimum level)
Weight loss 15%
Physical and
mental disability
Loss of
weight 7 to 15%

interstitial extracellular and passes water from the cells to the interstitial spaces, with secondary cellular dehydration (intracellular dehydration).

Syndromography or positive diagnosis

Clinical picture (fig. 69.3) and complementary

Clinically, the most important manifestation will be thirst, which appears early, since it is an index of intracellular dehydration. A center in the brain, probably located in the hypothalamus, is sensitive to this reduction in cell volume.

When the volume of secretions decreases, dryness of the skin and mucous membranes occurs; The tongue is dry and sticky, so when you place your finger on it, it does not slide, but rather sticks.

It is important to keep in mind that every patient with mouth breathing has a dry tongue; The tongue of the patient who is receiving oxygen also dries, even if he or she is washed. The uremic always has a dry tongue, even if its hydration is adequate.

The kidney eliminates highly concentrated urine; Oliguria with increased density is present and hyaline and granular cylinders are observed, which sometimes raise the question of the existence of nephritis, but this situation is normalized when the patient is hydrated.

In severe cases, profound cellular changes will occur, especially at the level of the brain, which explains the physical and mental alterations observed in these patients: asthenia, lack of energy, irritability; Sometimes stiffness of the neck.

Hyperthermia called dehydration fever may also occur, which is rare in adults.

As water continues to be lost, hemoconcentration gradually occurs, and the values of hematocrit, plasma sodium and other blood elements begin to rise proportionally;

It must be taken into account that if the patient is anemic, the hematocrit may show normal figures despite hemoconcentration. If the dehydrated person has abnormal amounts of non-electrolyte substances such as glucose (in a diabetic patient) or non-protein nitrogenous products (in a patient with nephropathy or liver disease) in the blood, the plasma sodium concentration may be normal, or only slightly increased, even when the water loss is marked.

The urine contains chlorides, sodium and the indicated casts.

Etiology (see Fig. 69.3)

Let us point out that, in general, dehydration is always due to:

- Decrease in water intake.
- Increase in aqueous losses.

Decrease in water intake

Any lack of supply of the 2,500 mL of water necessary to compensate for normal losses results in a predominance of fluid losses over income, which produces a state of dehydration, despite the fact that the body, in an attempt to save fluids, reduces the normal amount of urine.

As part of the water intake is obtained from solid foods, when a patient is kept on liquids, if they are given only what they normally consume, a state of dehydration can occur, since the liter of water that is provided with solid foods is missing. Absolute fasting naturally causes dehydration due to lack of intake. Any quantitative dietary restriction is, therefore, dehydrating.

Increase in aqueous losses

If we remember that normal losses take place: by evaporation, urine and faeces, any increase in evaporation (excessive sweating, fever or dyspnea), the amount of urine (polyuria due to diuretics, polyuric period of acute renal failure) and water loss with faeces (diarrhea), can lead to a state of dehydration. In addition to this, fluid losses due to vomiting or endogastric aspiration, states of ileus (mechanical paralysis), fistulas, etc., also lead, if not properly compensated, to a state of dehydration.

This type of hypertonic dehydration has been observed in:

1. Diabetes insipidus.
2. Cases of vomiting and diarrhea, with high temperature and great loss of water through the skin and lungs, or cases of insufficient water intake or when the patient is hydrated with solutions that have a high sodium content.
3. Comas, encephalitis, meningitis, with elevated temperatures and great loss of water through the skin and lungs.
nes.
4. Some acute respiratory conditions in children.
In general, in any pathological entity in which the final result is a loss of water greater than that of salts.
5. Cases in which foods that provide a large sum of solutes, such as carbohydrates, proteins, salts and other substances with an inadequate volume of water, are supplied to a patient through a Levine tube;
Here the water loss syndrome is due to excess solutes, which require more water for excretion than the body can supply.
6. Hyperosmolar coma.
7. Iatrogenic causes. It has been observed in children with gastroenteritis (vomiting and diarrhea) to whom mothers, considering that it is acidosis, administer sodium bicarbonate, therefore increasing plasma osmolality.

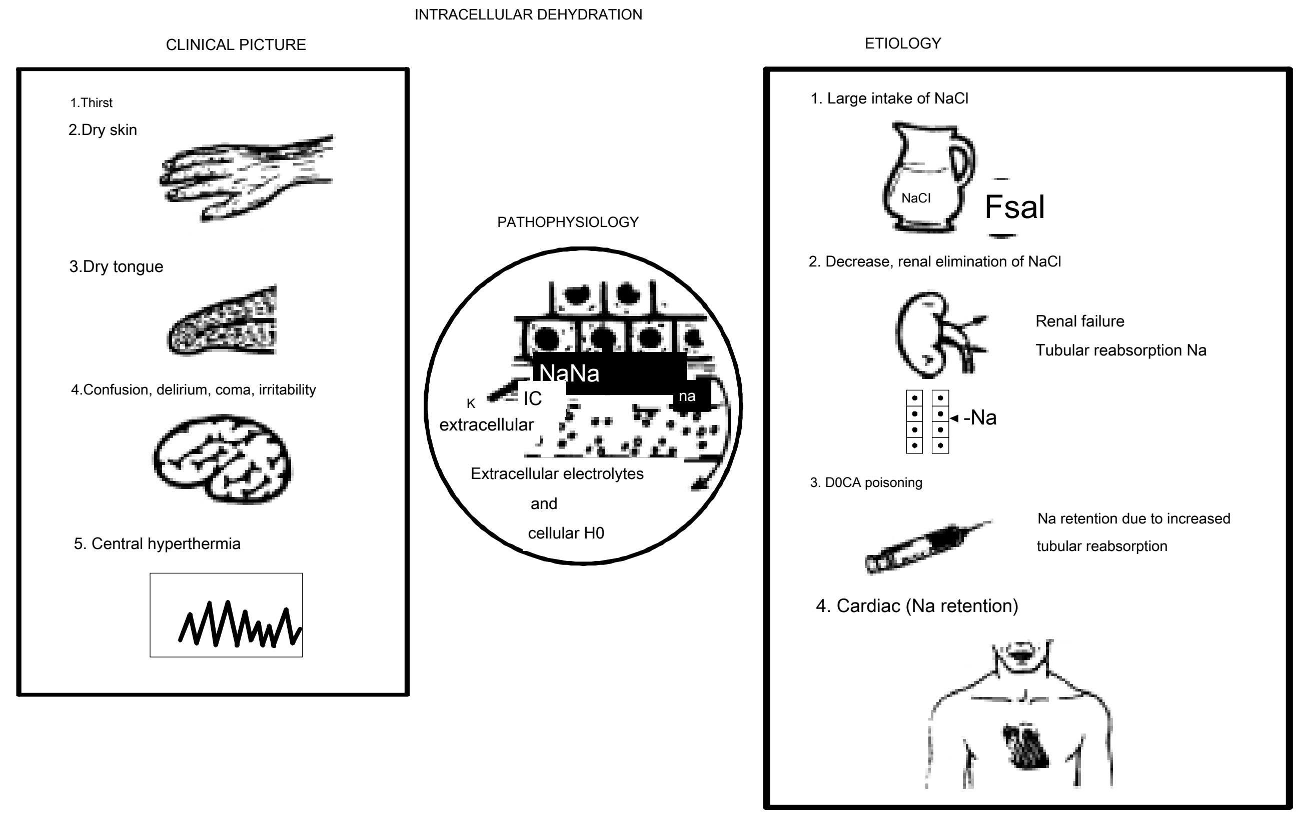

INTRACELLULAR DEHYDRATION
CLINICAL PICTURE
1.Thirst
2.Dry skin
3.Dry tongue
4.Confusion, delirium, coma, irritability
5. Central hyperthermia
PATHOPHYSIOLOGY
NaNa
na
K
IC
extracellular
Extracellular electrolytes
and
cellular H0
ETIOLOGY
1. Large intake of NaCl
NaCl
Fsal
2. Decrease, renal elimination of NaCl
Renal failure
Tubular reabsorption Na
-Na
3. D0CA poisoning
Na retention due to increased
tubular reabsorption
4. Cardiac (Na retention)

Fig. 69.3 Clinical picture, pathophysiology and etiology of hypertonic dehydration syndrome (cellular or intracellular dehydration).

Hypotonic dehydration syndrome

Synonymy

Secondary dehydration, extracellular dehydration, salt depletion syndrome, cellular hyperhydration syndrome.

Concept

This syndrome is characterized by the loss of water from the body but accompanied by a large loss of electrolytes.

This type of dehydration is seen in those patients who lose water and electrolytes due to diarrhea, vomiting, intestinal fistulas, etc., but only have their water replaced (5% dextrose).

It is also observed in adults who work in places with intense heat, which is why they sweat a lot and by ingesting plenty of water and eliminating it, this syndrome can occur.

Syndromogenesis or physiopathology

Hypotonic dehydration is determined by an alteration in the normal osmotic balance between the intracellular and extracellular fluid, in the sense that in tissue spaces, the osmotic pressure decreases due to impoverishment in electrolytes and as a result of this and to balance isotony, water will pass into the cell. This is, therefore, cellular hyperhydration due to extracellular osmotic hypotonia, with the consequent cellular swelling phenomena (fig. 69.4).

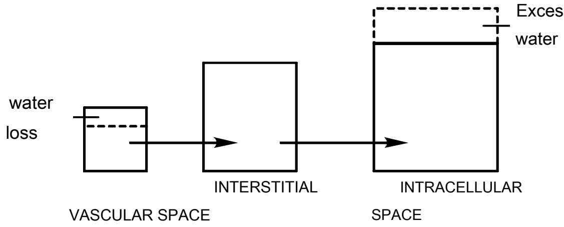

Excess
water
water
loss
INTERSTITIAL
INTRACELLULAR
VASCULAR SPACE
SPACE

Fig. 69.4 Hypotonic dehydration.

Syndromography or positive diagnosis

Clinical picture (fig.69.5)

Clinically, symptoms derived from cellular engorgement or turgor appear, especially in tissues in which greater cell integrity is required; For example, the system

I'm nervous.

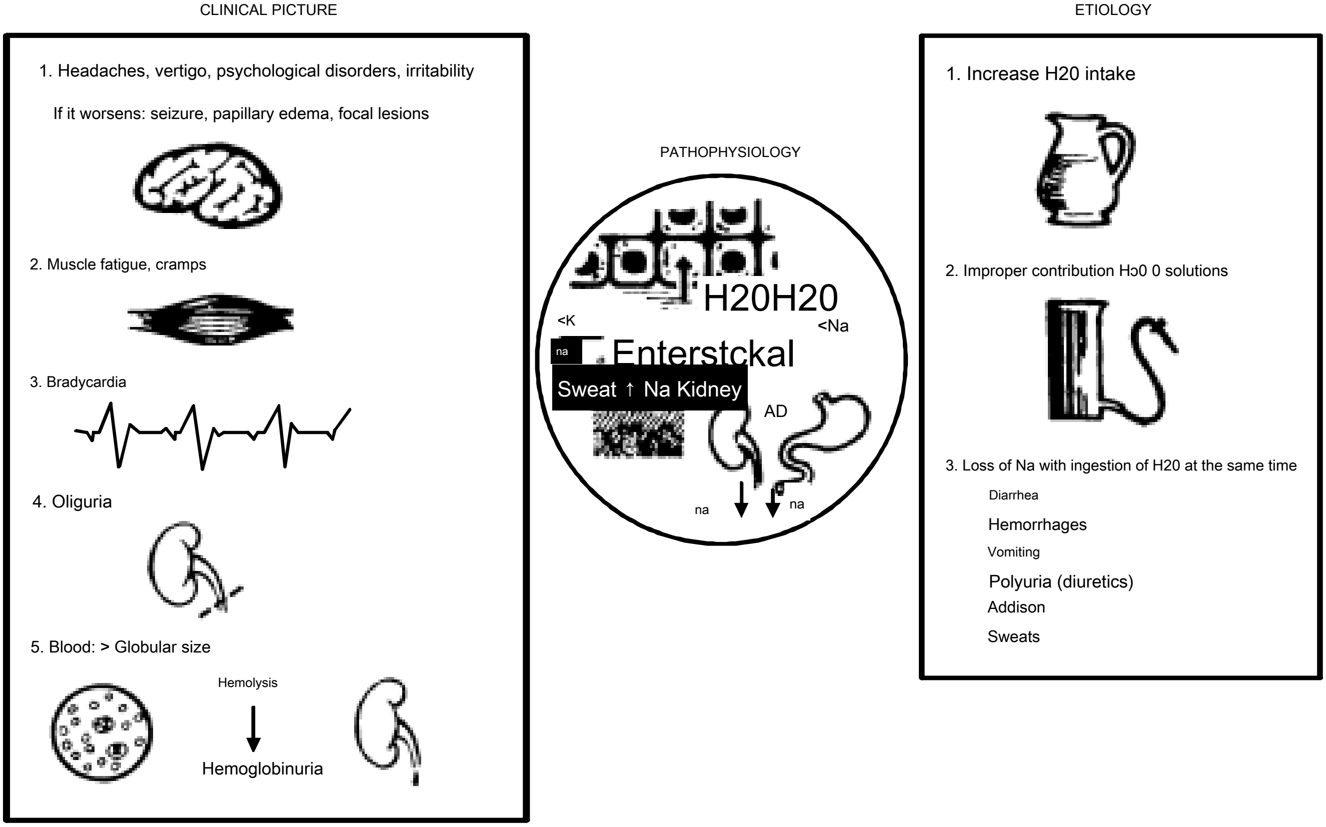

CLINICAL PICTURE
1. Headaches, vertigo, psychological disorders, irritability
If it worsens: seizure, papillary edema, focal lesions
2. Muscle fatigue, cramps
3. Bradycardia
4. Oliguria
5. Blood: > Globular size
Hemolysis
Hemoglobinuria
PATHOPHYSIOLOGY
H20H20
<K
<Na
na
Enterstckal
Sweat ↑ Na Kidney
AD
na
na
ETIOLOGY
1. Increase H20 intake
2. Improper contribution Hↄ0 0 solutions
3. Loss of Na with ingestion of H20 at the same time
Diarrhea
Hemorrhages
Vomiting
Polyuria (diuretics)
Addison
Sweats

Fig. 69.5 Clinical picture, pathophysiology and etiology of hypotonic dehydration syndrome (cellular hyperhydration).

The patient presents indifference, laxity and apathy; In the muscles, the feeling of intense asthenia is very common when attempting any physical exercise (very early symptom) and subsequently, the presence of cramps.

Dehydration causes contraction of the vascular compartment, due to hypovolemia, which explains the arterial and postural hypotension that these patients present and which in severe cases can lead to shock. The pulse is weak.

Anorexia is absolute for food and water; nausea and vomiting are evident; The latter are, moreover, the starting point of a vicious circle that aggravates osmotic hypotonia by exaggerating salt losses.

Nervous disorders are common, headache is an early sign and becomes more marked when the patient stands up. Dizziness, disorientation, psychological manifestations, visual disturbances are also observed and, if the syndrome continues to progress, ataxia, seizures, delirium and even coma will appear.

In severe dehydration the patient has a special appearance, difficult to describe, we can say that it is characterized by sunken eyes, or rather, by a dark circle around them; the patient is deeply “haggard”, the bone reliefs are They become more evident and the skin tightens a little, to the point of looking like a different person.

Dryness of the integuments with persistence of the skin fold represents its essential sign.

This sign, like that of dry tongue, should not be interpreted routinely. The elasticity of the skin is highly variable depending on the subjects and the areas that are explored. In the elderly it is greatly reduced, especially in the hands, and the positive fold sign is found very frequently in normal elderly subjects, especially if they are thin; Therefore, we must take into account the age of the patient for its evaluation.

The site of choice to explore this sign is the subclavicular space. In this area its appearance is, perhaps, a little later, but its value is much greater, since the local characteristics of the skin cannot give rise to error. However, we recommend exploring it in three or four sites to avoid local causes of error. Apart from the subclavicular space, the most suitable sites are the forearm, thigh and legs.

In general, the skin fold sign is a late sign that indicates, when it is evident, a high state of dehydration (loss of 20% of the total water in our body).

Examination of the peripheral veins of the hands can be useful for diagnosis; if the veins take more than

5s to fill, when the hands are below the plane of the right atrium, we must think of a decrease in blood volume.

Complementary exams

As glomerular filtration decreases, urea in the blood increases, there is an absence of chlorine and sodium in the urine, except in cases of Addison's disease or salt-losing nephritis. Plasma sodium is below 130 mEq/L, but we must remember to differentiate between true sodium depletion and dilutional hyponatremia.

Etiology (see Fig. 69.5)

The syndrome occurs when there is very repeated vomiting, repeated diarrhea, blood loss due to hemorrhages, polyuria (especially due to diuretics, which reduce the reabsorption of sodium, and, consequently, more of this cation is eliminated through the urine), and in severe states of diaphoresis or sweat elimination, as occurs in the tropics or in workers in high temperature places (ship stokers, etc.), since in all these cases a large amount of salt (NaCl) is lost and, as a consequence, the body becomes impoverished in sodium, making the condition worse due to the ingestion of water alone. Abuse of a salt-free diet can also cause it.

We can also include the states of cellular hyperhydration of severe adrenal insufficiency, nephropathies with oliguria or anuria and digestive saline losses (such as vomiting and diarrhea) that have already been mentioned above.

This type of dehydration is also seen in chronic adrenal insufficiency (Addison's disease), in certain clinical forms of adrenogenital syndrome, in salt-losing nephritis (chronic renal failure in which the tubule does not recover filtered sodium), in the indiscriminate use of diuretics, in the polyuric phase of acute renal failure, and, generally, in all conditions in which many electrolytes are lost and only the lost water is replaced.

Sometimes sodium is not lost, but is sequestered in the body (cases of intestinal obstruction, edema of burns or hypodermoclysis).

Table 69.1 shows the characteristics of the two types of dehydration studied, according to the predominance of water or salt loss.

Mixed dehydration syndrome

Synonymy

Isosmotic dehydration or global dehydration. It is the most frequently observed form in practice.

Concept

It is characterized by the joint losses of water and salts (due to vomiting, diarrhea, etc.), which ultimately reduce the three compartments. This type of dehydration can easily become hypertonic or hypotonic depending on the therapeutic approach followed (saline solution or 5% dextrose, for example).

Syndromogenesis or pathophysiology

In this syndrome, water and salts are lost; The loss of salts occurs because organic secretions

Table 691 Correlation of symptoms, according to the type of dehydration

Symptoms	Dehydration	
	(+++) Hypertonic water depletion (Primary or	(+) Hypotonic Sodium depletion (Secondary or extracellular)
Thirst		Absent
Laxity		+++
Postural hypotension	Absent	+++
Urine (NaCi)	often +	Absent except in Addison's disease and salt-wasting nephritis
Pulse	Normal, except at the end	Fast
Vomiting, cramps Plasma	Absent	They can be + + +
sodium Blood pressure	Slightly increased Normal, less at the end	Decreased +
		Very low
Skin fold Cause of	No	+++
death	Increased pressure Circulatory insufficiency	Peripheral osmotic circulatory insufficiency

son isotónicas; There are also obligatory daily losses (through the skin, lung, etc.) that are made up almost exclusively of water. This causes water to move from the intracellular to the extracellular compartment to compensate for the discrete extracellular hypertonia, creating dehydration of both spaces: intracellular and extracellular.

Syndromography or positive diagnosis

Clinical picture

It is caused by a mixture of the symptoms and signs of the two types of dehydration described above. Dryness of the tongue and skin is associated with a marked sensation of thirst.

If the condition persists, the skin fold sign, tachycardia, hypotension and collapse appear.

Complementary exams

Plasma sodium may be normal or elevated depending on water loss. Hemoconcentration increases hematocrit. In general, there are alterations in the acid-base balance. Urea may be elevated.

Etiology

It is observed in all patients who lose a large amount of secretions in the form of vomiting, diarrhea, etc., and do not ingest liquids. It is the type of dehydration that occurs in digestive fistulas, intestinal obstruction, diabetic coma, among others.

WATER IMBALANCE DUE TO EXCESS: HIPERHIDRATACIÓN

It includes the so-called hyperhydration syndrome.

We have already studied intracellular hyperhydration when describing the hypotonic dehydration syndrome, therefore, now we will describe the most common hyperhydration syndrome in the clinic.

Hyperhydration syndrome

Synonymy

Water intoxication, excess water syndrome or global hyperhydration.

Concept

It is characterized by an increase in extracellular and intracellular water, generally caused by iatrogenic behaviors.

Syndromogenesis or pathophysiology

It occurs whenever water is administered in a quantity greater than its maximum excretion rate by the normal or pathological kidney; also when there are hormonal alterations mainly in ADH.

Syndromography or positive diagnosis

Clinical picture

The patient presents hyperpnea, headache, nausea, vomiting, blurred vision (due to papilledema), disorientation, stupor and seizures. Most of these symptoms are due to intracranial hypertension.

In severe cases, acute lung edema may occur. It is common to find lacrimation, salivation and edema, as well as alterations in diuresis, which can lead to anuria.

Complementary exams

The blood count and hematocrit are low. Sodium, chlorine and plasma proteins decrease. Urea is normal.

Etiology

This syndrome occurs in:

1. Water administration in oligoanuria.
2. Water administration in the presence of increased ADH activity.
3. Administration of fast-drip glucose solutions during the postoperative period.
4. Heart failure.
5. Cirrhosis.

In summary, we can say that many times the syndromes described are associated in practice and give rise to different variants, which we have tried to represent in figure 69.6.

ALTERATIONS OF THE ELECTROLYTE BALANCE: BY EXCESS AND BY DEFECT

We have already seen the alterations in water metabolism that also involve electrolyte disorders, and that are often accompanied by alterations in the acid-base balance, so we must always keep in mind that, in general, isolated disorders do not occur but rather more than one sector is affected.

The study of hydromineral balance requires a measure of the eliminations, ingestions and dosage of the different electrolytes, as well as the study of the conditions that led the patient to that state. According to this, we are going to study:

- Chlorine imbalance.
- Sodium imbalance.

Potassium imbalance.

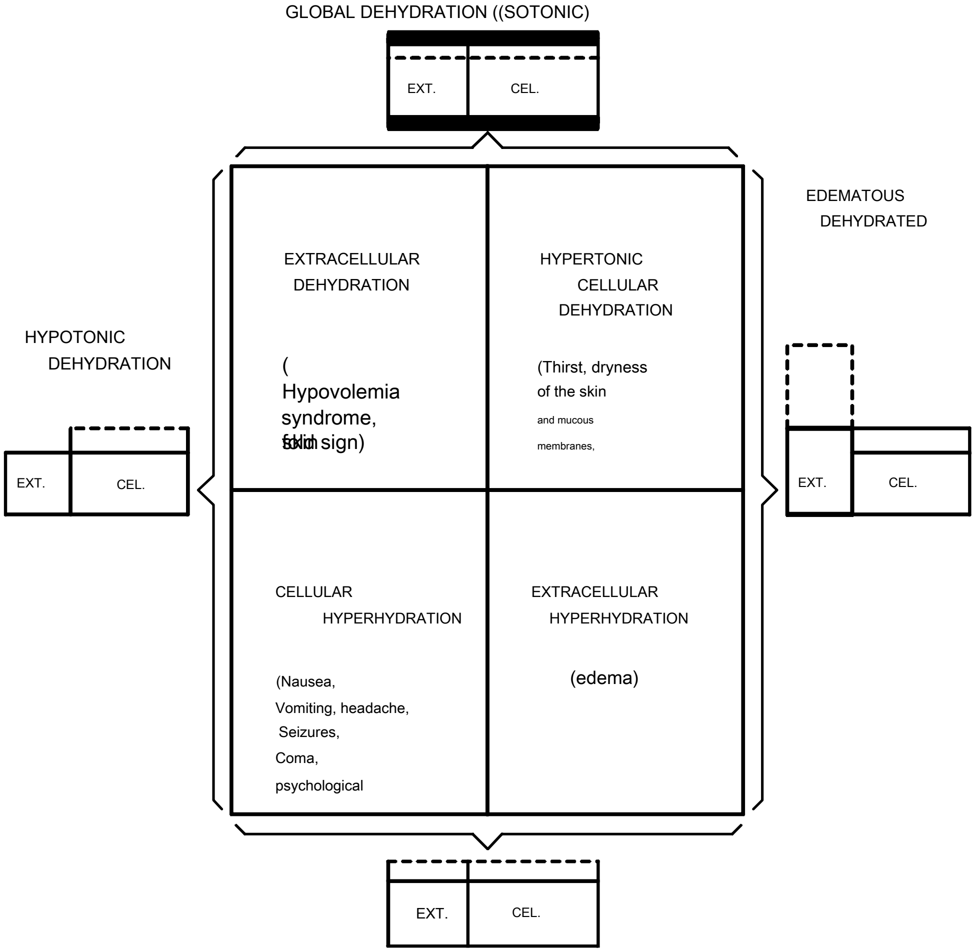

GLOBAL DEHYDRATION ((SOTONIC)
EXT.
CEL.
EDEMATOUS DEHYDRATED
EXTRACELLULAR DEHYDRATION
(Hypovolemia syndrome, skin fold sign)
HYPERTONIC CELLULAR DEHYDRATION
(Thirst, dryness of the skin and mucous membranes,
HYPOTONIC DEHYDRATION
EXT.
CEL.
EXT.
CEL.
CELLULAR HYPERHYDRATION
(Nausea, Vomiting, headache, Seizures, Coma, psychological
EXTRACELLULAR HYPERHYDRATION
(edema)
EXT.
CEL.

Fig.69.6 Clinical variants of water balance.

- Magnesium imbalance.
- Calcium and phosphorus imbalance.

CHLORINE IMBALANCE

It includes:

Hypochloremia syndrome.

- Hyperchloremia syndrome.

Hypochloremia syndrome

Syndromography or positive diagnosis

The two most striking symptoms are paralytic ileus and arterial hypotension.

Chlorine dosage completes the diagnosis, even if the ionogram is of no use, when it comes to dilutional hypochloremia, since it reports on low chlorine, without having been lost.

Etiology

1. Prolonged administration of salt-free diets.
2. Excessive elimination by the kidney (use of diuretics, nephritis).
3. Vomiting, aspiration or gastric fistula.
4. Dilutional hypochloremia.

Hyperchloremia syndrome

Syndromography or positive diagnosis

Thirst, tremors, confusion and sometimes fever occur. The history and the existence of significant hyperchloremia in the ionogram help in the diagnosis.

Etiology

1. Administration of chlorinated serum in the presence of renal failure. In these cases, unless

If the deficit is accentuated, such serums should not be administered.

2. Excessive supplies of salt in the diets of kidney patients.

SODIUM IMBALANCE

It includes:

-Hyponatremia syndrome.

- Hypernatremia syndrome.

Hyponatremia syndrome

Syndromography or positive diagnosis

The clinical picture is reminiscent of water intoxication: headache, dizziness, vomiting, hostile behavior and, in extreme cases, seizures and coma.

Pathophysiology and etiology

Three mechanisms control the body's sodium balance:

1. The mechanism of renal countercurrent.
2. Osmoreceptors in the hypothalamus that control ADH secretion.
3. The thirst mechanism, which maintains a sodium concentration of 138-142 mmol/L.

An increase in plasma sodium concentration is always accompanied by hypertonicity, but normal or decreased plasma sodium does not mean a euosmotic or hyposmotic state.

The presence of other osmotically active substances adds particles independent of the plasma sodium concentration, such as urea, mannitol and glucose.

If hyponatremia is hyposmolal, it is classified into three groups:

1. Hypovolemic hyponatremia.
2. Euvolemic hyponatremia.
3. Hypervolemic hyponatremia.

Hypovolemic hyponatremnia (with decreased total body sodium). The measurement of urinary sodium classifies them: if the sodium concentration in the urine is greater than 20 mmol/L, it is due to renal loss and those with less than 20 mmol/L, due to extrarenal loss.

Euvolemic hyponatremia. It is the most common cause of hyponatremia in the clinic. The urinary sodium concentration is above 20 mmol/L. It is seen in glucocorticoid deficiencies, hypothyroidism, antipsychotic drugs and in the syndrome of inappropriate ADH secretion. Drugs that cause hyponatremia are: thiazide diuretics; psychoactive agents, such as gleroxetine, haloperidol, amitriptyline and amphetamine; anticancer drugs, such as vincristine, vinblastine, cyclophosphamide, carbamazepine, and chloropropamide; and many other types of drugs.

Hypervolemic hyponatremia (with an increase in total body sodium, but a greater increase in total body water). This condition occurs in heart failure, liver cirrhosis, nephrotic syndrome, and all have a sodium concentration in the urine less than 20 mmol/L.

Hypernatremia syndrome

Syndromography or positive diagnosis

The symptoms of hypernatremia depend on plasma hyperosmolality and have already been considered when talking about hypertonic dehydration.

Classification

a) With lower total sodium and lower total body water.

b) With an increase in total sodium and total water.

c) With normal total sodium and decreased total body water.

Syndromogenesis and etiology

a) Hypernatremia with lower total sodium and lower total body water.

It is due to two causes: renal losses or extrarenal losses.

In renal losses, the sodium concentration in urine is greater than 20 mmol/L (diuretics, post-obstructive diuresis, interstitial nephritis).

In extrarenal losses, the sodium concentration in urine is less than 20 mmol/L (copious sweating, febrile syndromes, burns, diarrhea, enteric fistulas).

b) Hypernatremia with increased total sodium and increased total body water occurs with the administration of hypertonic sodium (3%) or with the administration of large amounts of sodium bicarbonate, in the event of metabolic acidosis or cardiorespiratory arrest, in hyperaldosteronism and in Cushing's syndrome.

In these cases, the measurement of urinary sodium is always above 20 mmol/L.

c) Hypernatremia with normal total sodium and decreased total body water. There are two groups of causes: one, extrarenal, due to insensible respiratory and skin losses, such as in febrile and hypermetabolic states, and the other, due to renal losses, such as diabetes insipidus and hypodipsia. The urinary sodium concentration in these cases does not serve to differentiate.

POTASSIUM IMBALANCE

Potassium regulation has an acute phase of distribution between the intra- and extracellular spaces and a chronic phase of renal regulation.

In hypokalemia, less than 15 mmol/day of potassium is excreted, and in hyperkalemia, more than 200 mmol/day.

If potassium excretion is abnormal, according to its serum concentrations, it is necessary to separately examine the two components of potassium excretion:
the urine flow in the collecting duct and the transtubular potassium gradient.

The urine flow in the collecting tube is increased when it is greater than 2 L/day.

The transtubular potassium gradient (TTKG) is the relationship between the potassium concentration in the lumen of the cortical collecting duct ([K]en) and the potassium concentration in the peritubular capillaries or in the plasma ([K*]).

$$\text{TTKG} = \frac{[K^*}{[K^*]ccp} \qquad (1)$$

The validity of this calculation is based, among other things, on the fact that potassium is not secreted or reabsorbed in the medullary collecting duct (MCT) and that the Osmolality of the fluid in the terminal cortical collecting duct (Oosm) is known.
So:

$$[K^*Jocp = \quad [K^*l, : \quad \frac{Oosm}{posm} \qquad (2)$$

When vasopressin (antidiuretic hormone) is acting, the osmolality in the proximal cortical collecting duct (CCD) is the same as that of plasma and the potassium concentration in the lumen of the distal cortical collecting duct (Oosm/Posm= 1).

To calculate TTKG, urine osmolality must exceed that of plasma.

Substituting (2) into (1) the final formula for the transtubular potassium gradient (TTKG) is:

$$\text{TTKG} = [K^*J, : \quad \frac{Oosm}{posm} \quad [K^*], \; (3)$$

It involves dividing the concentration of potassium in urine ((K1) by the result of dividing the osmolality of the urine (Oosm) by the osmolality of the plasma (Posm), and all this, divided by the concentration of potassium in the plasma [K‘,

Significant potassium reabsorption or secretion in the TCM rarely occurs except in severe potassium depletion or excess, respectively.

There is no normal range of values for the transtubular potassium gradient (TTKG), since it depends on the balance state of the entire body's potassium. A TTKG greater than 2 suggests renal potassium loss due to increased distal secretion of potassium, usually due to excess mineralocorticoids (hyperaldosteronism). When the TTKG is less than 2, it is due to a mineralocorticoid deficiency.

The clinical study of potassium imbalance includes two syndromes:

Hypokalemia syndrome.

- Hyperkalemia syndrome.

Hypokalemia syndrome

Syndromography or positive diagnosis

Clinical picture

It is based on the following symptoms:

1. Asthenia and muscle weakness that can lead to paralysis; By palpation we check soft muscles. Tendon reflexes are decreased.
2. Paralytic ileus and bladder atony in severe cases.
3. Sometimes tremors occur.
4. Postural hypotension and in severe cases, peripheral vascular collapse.
5. Cardiac disorders, since myocardial necrosis similar to those seen in cases of digitalis poisoning can occur.

Complementary exams

1. ECG: Flattened or inverted T wave, lengthened QT and appearance of the U wave.
2. Severe hypokalemia can stop the heart in systole by increasing myocardial irritability.
3. Extracellular alkalosis that can lead to tetany.
4. Kaliopenic nephropathy occurs at the kidney level, which is characterized by vacuolization of the proximal tubule and is manifested by polyuria, polydipsia and loss of concentration function. At the beginning there are no filtration alterations and therefore, there is no nitrogen retention. These alterations are similar to those observed in primary hyperaldosteronism or Conn syndrome.

As we already said, hypokalemia with a transtubular potassium gradient (TTKG) greater than 2 suggests an excess of mineralocorticoids (hyperaldosteronism).

Plasma levels of renin and aldosterone often help differentiate the various causes of hyperaldosteronism. Bicarbonaturia and the presence of other non-reabsorbed anions also increase TTKG and lead to renal potassium loss.

Etiology

1. Potassium depletion.
2. Dilutional hypokalemia.
3. Displacement of extracellular fluid into the cells: alkalosis, administration of glucose plus insulin and periodic familial paralysis.

Among the causes of potassium depletion, we have: vomiting and diarrhea, Cushing's syndrome, administration of corticosteroids, primary hyperaldosteronism, administration of diuretics, polyuric phase of acute tubular necrosis and suppurations, especially abdominal.

Hyperkalemia syndrome

Syndromography or positive diagnosis

It is vague and confusing, the symptoms of the causal condition predominate. Some cases may present: progressive asthenia, which sometimes reaches paralysis and cramps and diarrhea.

Among the complementary tests, the most important are the electrocardiographic manifestations that appear when potassium exceeds 7 mEq/L: high, narrow and pointed T wave; shortening of QT and disappearance of ST, with wider QRS; lengthened PR interval and, finally, disappearance or inversion of the P wave.

In severe cases there is an idioventricular rhythm, which paralyzes the heart in diastole.

Etiology

1. Acute and chronic renal and adrenal insufficiencies.
2. Acidosis.
3. When there is severe tissue damage due to burns or crush syndrome, although in these cases, if kidney function is not affected, potassiumuria prevents hyperkalemia.

MAGNESIUM IMBALANCE

It includes:

- Hypomagnesemia syndrome.
- Hypermagnesemia syndrome.

Hypomagnesemia syndrome

Syndromography or positive diagnosis

The clinical diagnosis of hypomagnesemia is difficult, since it is usually accompanied by other electrolyte deficiencies.

The main manifestations described are neurological: lethargy, muscle weakness, fasciculations, tremors, athetoid movements, convulsions, tetany and eat.

Overt tetany is rare and when it occurs it is usually due to alkalosis or hypocalcemia. The nausea, vomiting and anorexia that sometimes accompany hypomagnesemia aggravate the clinical picture. Cardiac arrhythmias have also been described.

Sometimes there is magnesium depletion without a drop in serum levels, but from a practical point of view this information is important to have an idea of the magnesium balance.

Etiology

1. Liver cirrhosis.
2. Intestinal malabsorption syndrome.
3. Use of diuretics (especially in heart failure).
4. Alcoholism.
5. Pancreatitis.
6. Diuretic phase of acute tubular necrosis.
7. Diabetic acidosis.
8. Hypoparathyroidism.
9. Primary aldosteronism.

Hypermagnesemia syndrome

Syndromography or positive diagnosis

The main manifestations are: lethargy, coma, accentuation of renal failure, decreased patellar reflex, depression of the nervous system, cardiac conduction disorders, etc.

Etiology

They are rare. They are observed in oliguric uremic patients who are administered antacids (magnesium trisilicate) or magnesium sulfate.

CALCIUM AND PHOSPHORUS IMBALANCE

It includes:

- Hypocalcemia syndrome.
- Hypercalcemia syndrome.

Hypocalcemia syndrome

Syndromography or positive diagnosis

Among the symptoms of hypocalcemia we have tetany, tingling of the lips, abdominal and muscle cramps and hyperreflexia. Calcium and phosphorus measurements corroborate the diagnosis.

Etiology

1. Hypoparathyroidism.
2. Pancreatitis and peritonitis.
3. Malabsorption syndrome.
4. Duodenal and pancreatic fistulas.
5. Hypovitaminosis D (rickets).
6. Excessive administration of citrated blood.
7. Pseudohypoparathyroidism. In this case the parathyroid is normal, although there is hypocalcemia that does not respond to the parathyroid hormone; We see that when injecting this, phosphates are not eliminated, since the tubule does not respond to the hormone. It is, therefore, a pseudoendocrinopathy due to nephropathy. It is accompanied by oligophrenia and short stature: hypoparathyroid cretinism.
8. Chronic kidney disease. In these cases there is hypocalcemia due to several causes:
    - a) Phosphates are retained in the blood, therefore calcium decreases.
    - b) Sometimes there is distal tubular insufficiency, NH is not formed (renal acidosis). In this case, acidosis causes calcium to be mobilized from the bones (basic ion), and nephrocalcinosis may occur due to the large elimination of calcium.
    - c) Lack of absorption of vitamin D by the intestine.

Both hyperphosphatemic processes (kidney lesions, hypervitaminosis D, hyperchlorhydria, diets rich in fat and low in Catt) and hypocalcemic processes are accompanied by secondary hyperparathyroidism.

Hypercalcemia syndrome

Positive diagnosis, etiology and clinical picture

In hypercalcemia there is muscle relaxation, which causes fatigue, weakness and hypotonia of the muscles, as the main symptoms.

It is observed in hyperparathyroidism, whose most typical clinical picture is Recklinghausen's disease or osteitis fibrosa cystica, in which the patient presents bone cysts with pain, osteoporosis, tendency to fractures, muscle hypotonia and kidney stones.

Hypercalcemia is also present in iatrogenic hypervitaminosis D, a clinical condition that is characterized by weakness, headache, dizziness, uremia due to nephrocalcinosis and sometimes, soft tissue calcification.

Finally, we observe hypercalcemia accompanied by hypercalciuria and hypophosphatemia, in osteolytic metastases, multiple myeloma, multiple fractures and in sarcoidosis.

CHANGES IN THE ACID-BASE BALANCE

For a long time there was confusion about the acid-base balance, since anions were called acids and cations were called bases; However, the bicarbonate ion (HCO,), which is the base par excellence, is in the anion column.

An acid is a donor of H, for example, HCl.
A base is an acceptor of H for example, HCO,-.

We call strong acids or strong bases those components that dissociate completely into their component ions in a solution.

The potency of an acid or base is measured by the degree to which it dissociates, and the concentration of Ht or OH ions it contains.

A weak acid is one that dissociates little, therefore, its hydrogen concentration does not increase, for example, H,CO,.

Under normal conditions, the pH of the blood remains between 7. 35-7.45, despite the large amount of acids and bases that are constantly being produced in the body as a product of tissue metabolism.

In pathological conditions the pH can vary between figures of 6.8-7.8. Figures that exceed these limits are incompatible with life.

REGULATION OF ACID-BASE BALANCE

To maintain the concentration of hydrogen ions and the pH within normal limits, the body uses a series of regulatory mechanisms, among which we have:

1. Blood buffers.
2. Lung regulation.
3. Kidney regulation.
4. The exchange of ions between the different compartments.
5. Decarboxylase enzymes.

We understand by buffers, tampons or shock absorbers, those chemical substances capable of reacting as acids or as bases depending on the circumstances. These substances have the property of reducing sudden changes in pH that may occur when adding a strong acid or alkali to a solution.

Buffer systems result from the mixture of a weak acid and its alkali salt, or a weak base and its acid salt.

In the body, the buffers of physiological importance are mixtures of weak acids and their alkaline salt; For example:

$$\frac{\text{NaHCO,}}{\text{H,CO,}}$$

The alkaline salt of bicarbonate (NaHCO,) has a sodium cation (Na) and a bicarbonate anion (HCO,) and gives up its cation to any strong acid that penetrates the organism (HCl, for example), to form a weak acid (H,CO.), which dissociates little and does not increase, therefore, the hydrogen concentration. It is a reserve base that we have in the body to react with any strong acid that is produced. For this reason we call the bicarbonate ion alkaline reserve.

NaHCO, + HCl NaCl + H,Co,

By virtue of this reaction, the pH or acidity is not altered, because thanks to it a strong acid (HCl) is exchanged for a weak one (H,C0,). All tampons have this advantage.

The most important buffer of the extracellular fluid is bicarbonate (HCO,), not only because it is the most abundant in the body, but also because its conjugate acid, carbonic acid (H,CO,) has the advantage of quickly disintegrating into H,0 and CO,, a gas that is immediately eliminated by the lung and can be independently controlled by the respiratory function.

Due to these advantages of bicarbonate, the HCO,/H,C0 relationship is the one that interests us the most:

$$\frac{\text{HCO,}}{\text{H,CO,}} \qquad \frac{\text{HCO,}}{\text{CO, diluted}} \qquad \frac{20}{1}$$

As long as this relationship remains constant, the pH will not vary, even if the numerator or denominator increases or decreases proportionally.

HCO is measured indirectly in volumes of CO.

The normal figure is 55-69 volumes of CO,.

The bicarbonate-carbonic acid system corresponds to 53% of this total buffer action.

Another blood buffer, which operates mainly in the kidneys as we will see later, is the phosphate system:

$$\frac{\text{Na,HPO,}}{\text{NaH,PO,}}$$

The protein system constitutes another blood buffer, represented by the relationship:

$$-\frac{\text{proteinate}}{\text{protein}}\ .$$

Proteins are amphoteric substances, that is, they ionize depending on the circumstances, releasing in some cases H, and in others OH radicals, which as we know are hydrogen ion scavengers. When there is an acidosis, they ionize as alkalis, releasing OH, and when the overload is bases, they ionize as acids, releasing H*.

Through the hemoglobin system, the chlorides of the plasma are exchanged for the bicarbonate of the red blood cells;
This system constitutes 35% of the body's total buffer effect.

We will describe the other mechanisms that the human body uses to maintain pH within normal limits in the study of acidosis and alkalosis.

DEFINITIONS OF ACIDOSIS AND ALKALOSIS

Acidosis or alkalosis are those situations in the body in which the acid-base balance and the general pH of the internal environment, externalized by the blood, tend to alter within the normal figures of 7.4 towards the alkaline side or towards the acidic side. The acidosis syndrome is also called acidemia, a term that encompasses greater specificity with respect to blood acid-base alteration; alkalosis is also usually designated by other authors as alkalenmia.

There is still a state called ketosis, which is actually an acidosis, but its cause lies in an increase in certain fatty acids in the blood. Within these syndromes we must distinguish the concepts of compensation and non-compensation.

It is possible to speak of compensated acidosis and alkalosis when, despite the humoral and clinical disorder that may occur, the blood pH remains normal. These cases indicate that there is compensation by the pH regulatory mechanisms and that this regulation itself can disturb the normal physiology and health of the patient, although not the true pH, which remains at its normal constant. On the contrary, uncompensated states are those in which the pH enters in the pathological figures (less than 7.3 for acidosis states and greater than 7.4 for alkalosis states).

LABORATORY STUDIES ON ACID-BASE IMBALANCE

Hemogasometry

Laboratory diagnosis of alterations in the acid-base balance has become more accessible and much more accurate since 1962, when Paul Astrup and his collaborators, taking advantage of the pressure of the glass microelectrode designed by Sanz, made the microgasometric method available to doctors.

Using this method, pH and pCO are quickly determined; bicarbonate can be deduced using the curved nomogram of Siggaard, Andersen and Engel.

Through a series of uncomplicated operations, adequate parameters can be obtained that allow determining any type of alteration of the acid-base balance, although in the case of metabolic acidosis it is not possible to determine the type of acid in question responsible for the imbalance. For example, a patient may be in metabolic acidosis, but it cannot be determined whether it is hyperlactacidemia or an increase in beta-hydroxybutyric acid or ketoacetic acid.

Below we will describe each of the parameters obtained by gasometry.

pH

It is the negative logarithm of the hydrogen ion concentration. Lately, there is a tendency to use terminology comparable to that of electrolytes, to refer to pH; However, the milliequivalent unit (mEq) is not practical due to the excess of decimals and in order to speak in positive terms, the millimicroequivalent or nanoequivalent (nEq) is used, as will be seen later).

Normal values

Arterial and capillary blood: 7.35-7.45.

Venous blood: 7.28-7.35.

Partial pressure of CO, (pC0,)

Carbon dioxide concentration in the blood analyzed:

Normal values

Arterial and capillary blood: 35-45 mm Hg.

Venous blood: 45-53 mm Hg.

Base buffer (BB)

It represents the total buffering potential of blood, including bicarbonate, serum protein, hemoglobin, phosphates and others.

Bicarbonate assumes approximately 50% of this potential buffer. It is the most mobile and faster action; By the Astrup method it is called standard bicarbonate (BS).

Normal values

in arterial blood and venous blood: 42-50 mEq/L.

Standard bicarbonate (BS)

It is the concentration of bicarbonate present in the plasma, when the blood has been subjected to complete oxygenation of the hemoglobin, the pCO, is 40 mm Hg and the temperature is 37 °C.

Normal values

in arterial blood and venous blood: 21-25 mEq/L.

Base excess (BE)

It is the amount of strong acid or base in mEq/L that must be added to plasma or blood to reach a pH of 7.4 when lapCO is 40 mm Hg, at a temperature of 37 °C.

The amounts of strong acid or base to be added are determined by titration. If the pH is less than 7.40, strong bases (NaOH) must be added. The milliequivalents of NaOH or HCl expended in the titrations, per liter of plasma or blood, constitutes the EB value.

The EB can be used to measure the magnitude of the deviation from normal of the total buffer system. The EB is equal to the difference between the normal BB minus the patient's BB.

The EB measures both the excess and the deficiency of bases or non-volatile acids. It is the value corresponding to the " net" accumulation of fixed acid or base in plasma or whole blood.

This value says nothing as to whether a simultaneous accumulation of acids or fixed bases has taken place, nor what kind of acids or fixed bases are responsible for the situation.

Normal values: correspond to + 2.5 mEq/L, both in venous and arterial blood.

The alteration of each hemogasometric parameter, its production mechanism and its diagnostic significance are shown in Table 69.2.

In addition to gasometry, it is necessary to perform other laboratory tests that allow obtaining the fundamental parameters for a complete study, as we will see below.

Table 692 Alterations of hemogasometric parameters in acid-base imbalances

Parameter	Definition	Diagnosis
pH (low)	Acid excess or base deficiency	Total acidosis
pH (high)	Base excess or acid deficiency	Total alkalosis
PCO, (high)	Excess CO, CO	Respiratory acidosis
PCO, (low)	deficiency,	Respiratory alkalosis
EB (decreased)	Excess non-volatile acids or deficiency of bases	Non-respiratory acidosis
EB (augmented)	Excess of bases or deficiency of non-volatile acids	Non-respiratory alkalosis
BS (bass)	Excess of non-volatile acids or deficiency of bases	Non-respiratory acidosis
BS (high)	Excess of bases or deficiency of acids non-volatile	Non-respiratory alkalosis

Fundamental parameters for the study of acid-base balance

The four determinations required for its complete study are:

1. Concentration of hydrogen ions ([H) in blood.
2. PCO, blood.
3. Bicarbonate (HCO,).
4. Calculation of remaining anions (RA) from the serum ionogram.

These parameters and other necessary parameters, including the normal values of hemogasometry and ionogram, are summarized in Table 69.5. We will discuss some of them separately.

Hydrogen ion concentration [H]

The concentration of hydrogen ions in body fluids has traditionally been expressed by pH, which is the negative logarithm of [H], as was already seen in the study of the components of hemogasometry, but increasingly laboratories give the direct measurement in mEq/L, or better, in nanoequivalents per liter (nEq/L). This is because its use seems more logical, since the value increases to the same extent as the degree of acidosis, while the pH decreases. Furthermore, in some disorders such as respiratory acidosis, there is a calculable linear relationship between the values of [H] and blood PCO, while the relationship between the latter value and the pH is not linear. Finally, given two of the three variables (H] in nEq/L, PC0, in mm Hg and HCO, in mEq/L), the The third can be quickly calculated by applying a simple formula, derived from the Henderson-Hasselbalch equation, which is as follows:

$$[H+] = 24 \cdot \frac{PCO,}{HCO,}$$

The formula is also useful to correct possible laboratory errors, since the three values, when they are exact, must satisfy the equation, and to calculate [H1 when one of the other two variables is modified in the clinic, for example, the PCO, of a patient connected to a respirator.

The normal value of [H] is 40 nEq/L (for a pH of 7.40).

It is interesting and useful to know that in the pH range between 7.20 and 7.50, the sum of the last two digits of the pH with the value of [H] is equal to 80.

Examples:

for a pH of 7.35 1la [H] is 45 nEq/L (35 + 45_ = 80);

for a [H] of 38 nEq/L, the pH is 7.42 (38 + 42=80).

That's why,

for a pH = 7.40, the [H] = 40 nEq/L (40 + 40 = 80).

pH values of 7.20 or lower and 7.50 or higher do not coincide with this sum.

Table 695

Fundamental parameters for the study of acid-base balance disorders.

Arterial or capillary blood gases:

pH	7.35-7.45 (45-35 nEq/L)
PCO.	35-45 mm Hg
HCO,	26 + 2 mmol/L
PO,	> 80 mmHg

H+ concentration using the Henderson-Hasselbalch equation:

$$[H] = 24. \frac{PCO,}{HCO,}$$

Ionogram:

Nat	140 mmol/L
K+	4.2 mmol/L
c	106 mmol/L

HCO,= 26 + 2 mmol/L

Remaining anions (AR):

$$AR = Na^* - (CH + HCO,$$

Calculated plasma osmolality (0SM,) (in mOsm/kg water)

$$2 (Na^* + K^+) + \frac{\text{glucose (mg \%)}}{18} \quad \frac{\text{urea (mg \%) 6}}{}$$

$$12 (Na^* + K^*) + \text{glucose (mmol/L)} + \text{urea (mmol/L)}$$

Osmolal gap = 0SM, measured - OSM, calculated = 8 mOsm/kg water

PCO, blood and bicarbonate calculation

As expressed previously, both values can be calculated or confirmed, from the formula, knowing two of the three variables. Blood PCO can be obtained from hemogasometry, while bicarbonate values are also expressed in relation to the study of the bases in it or are obtained from the serum ionogram.

Metabolic acidosis and alkalosis are primary alterations of plasma bicarbonate concentration, while respiratory disorders (acidosis and alkalosis) are primary alterations of PC0,. Table 69. 3 summarizes the primary alterations and compensatory changes of acid-base balance disorders.

Remaining anions (AR)

They are also known as innominate anions, anion gap, anion gap, R radical and gap anion.

They are calculated from the plasma values of sodium, chloride and bicarbonate, from the ionogram or, in the case of bicarbonate, the figures provided by other laboratory tests, as we have already seen.

It constitutes an important parameter for the diagnosis of metabolic acidosis. Its calculation is based on the fact that the sum of the cations (Nat + K) is greater than that of the anions (Cl + HCO,). Since the sum of the plasma anions must be equal to that of the cations, the remaining or unnamed anions that are not measured are made up, under normal conditions, of negatively charged plasma proteins, fundamentally albumin, and to a lesser extent by anions of organic acids, such as lactate, phosphates and sulfates. As the concentration of plasma potassium moves within very narrow limits and is very low compared to that of the other electrolytes measured, it is most often suppressed and the final formula for the calculation is:

AR calculation example:

If Na= 140 C=106 and HCO,- = 26

Substituting into AR = Na -(Ct + HCO,) AR = 140 - (106 + 26)

AR = 140 -132

AR = 8 mmol/L

Its normal value is 12 + 2 mEq/L.

Table 623 Primary alterations and compensatory mechanisms of acidosis and alkalosis

Flaw	At primary teration	Compensation	$\frac{PCO,}{HCO,-}$
Respiratory acidosis	CO retention,	HCO generation,	$\frac{PCO,}{HCO,}$
Respiratory alkalosis	Depletion Co,	HCO consumption,	$\frac{PCO,}{HCO,}$
metabolic acidosis	HCO Depletion,-	↑ Ventilation	$\frac{PCO,}{HCO}$
metabolic alkalosis	HCO retention,	J Ventilation	$\frac{PCO,}{HCO,}$

Classification of balance disorders acid-base

Before studying each of the syndromes and with the elements for the study of alterations in the acid-base balance that we have expressed, we must know how to classify the disorder, in such a way that we can act accordingly, for its correction.

Acid-base balance disorders can be simple, which are the four we have mentioned so far; mixed, when there is a combination of two of these simple disorders; and triple, when three of the four possible simple disorders coincide (Table 69.6).

Expected compensation ranges in simple disorders

The compensation ranges expected in the diagnosis of the four simple disorders of acid-base balance are expressed in Table 69.4.

acidosis syndrome

Acidosis exists whenever there is an excess of acids in the blood or a deficiency of bases. In acidosis there is always an increase in the concentration of hydrogen ions [H] in the extracellular fluid.

There are two types of acidosis: metabolic, which occurs when there is a primary metabolic disturbance, and respiratory, which is determined by primary respiratory conditions.

metabolic acidosis

Concept

In these cases, acidosis occurs due to an excess of strong acids or a primary deficiency of basic ions in the extracellular fluid. It is the most common type of acidosis in the clinic,

Syndromogenesis or pathophysiology

Metabolic disorders determine an increase in acid ions, as occurs, for example, in decompensated diabetes mellitus, in which the presence of certain acids such as diacetic and beta-hydroxybutyric acids give rise to a large production of hydrogen ions. This disorder can also occur when basic ions are lost, as occurs in diarrhea, for example, in which there is a large loss of bicarbonate.

As a respiratory compensation mechanism, a polypnea occurs that tends to eliminate CO2. The production of hydrogen ions represents the primary alteration and polypnea, with elimination of C0, (from H,CO,), a secondary compensatory mechanism.

This secondary mechanism tends to reduce the concentration of carbonic acid in order to maintain the bicarbonate/carbonic acid ratio at 20/1 and thus stabilize the pH. When this mechanism is not sufficient, the pH drops and we are in the presence of a decompensated acidosis.

Table 69.4 Expected compensation ranges in simple alterations of acid-base balance

Primary defect	Compensation	Ranks
metabolic acidosis	pCo, J.	pCO, = 1.5 (C0,H) + 8 + 2
metabolic alkalosis	pCo, ↑	pCO, = 0.9 (HCO.) + 9
Respiratory acidosis	HCO,-↑	Acute 1 mEq/L HCO, for every 10 mm Hg of pCO, Chronicle 3.5 mEq/L HCO, for every 10 mm Hg of pCO,
Respiratory alkalosis	H.C.O., J.	Acute 2 mEq/L of HCO,- for every 10 mm Hg of pCO, Chronicle 5 MEq/L of HCO,- for every 10 mm Hg of pCO,

Table 696

CI complete assimilation of acid-base balance disorders

Simple disorders

Respiratory:

Respiratory acidosis (acute and chronic)
Respiratory alkalosis (acute and chronic)

Metabolic:

Metabolic acidosis Metabolic alkalosis

Mixed disorders

Respiratory and metabolic:

Respiratory acidosis + metabolic acidosis Respiratory acidosis + metabolic alkalosis Respiratory alkalosis + metabolic acidosis Respiratory alkalosis + metabolic alkalosis

Mixed metabolic:

Metabolic acidosis + metabolic alkalosis Acidosis With traces of anions + hyperchloremic acidosis

Mixed acidosis with traces of anions
Mixed hyperchloremic acidosis

Triple disorders

Metabolic acidosis + metabolic alkalosis + respiratory acidosis Metabolic acidosis + metabolic alkalosis + respiratory alkalosis

The primary defect of acid-base disturbance is diagnosed by the clinical picture and mixed defects, by the separation of the compensation range of the primary defect.

Very important is the renal compensation function that tends to increase the excretion of the H ion by the renal tubular cells, which is exchanged for Nat so that it returns to the extracellular spaces in the form of NaHCO, which in turn tends to neutralize acidosis.

In these cases the urine will be acidic (fig. 69.7).

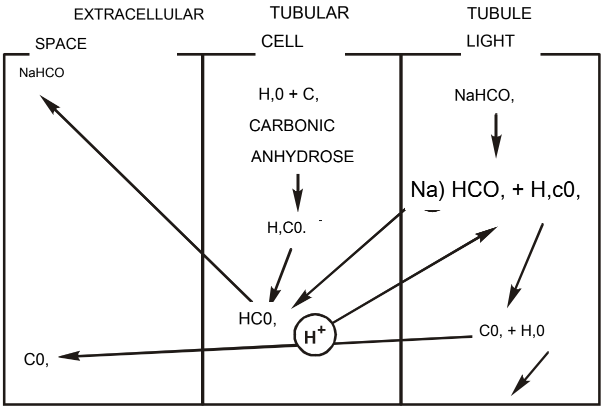

EXTRACELLULAR
SPACE
TUBULAR
CELL
TUBULE
LIGHT
NaHCO
H,0 + C,
CARBONIC
ANHYDROSE
H,C0.
NaHCO,
Na) HCO, + H,c0,
HC0,
H+
C0, + H,0
C0,

Fig. 69.7 Renal compensatory mechanism in metabolic acidosis. NaHCO bicarbonate pathway.

Furthermore, large proportions of Ht are excreted in the distal tubule, which is exchanged for Na obtained from Na, HPO, transforming it into NaH,PO,. The remaining Nat returns to the blood as bicarbonate (Fig. 69.8).

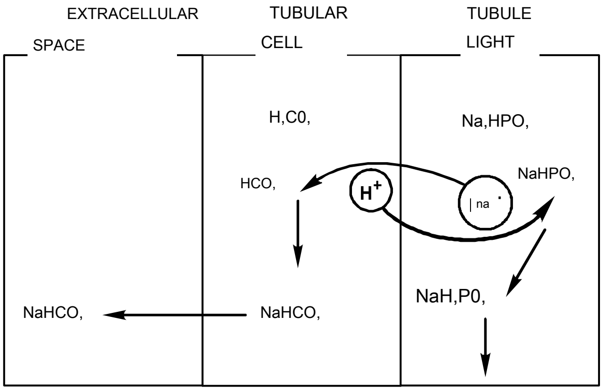
Finally, the secretion of ammonia by the tubular cells increases, which is exchanged with salts of strong acids (NaCl, for example), thus saving bicarbonate and excreting ammonium chloride through the urine (fig. 69.9).

To try to correct a metabolic acidosis, other buffer mechanisms already mentioned also intervene.

Syndromography or positive diagnosis

It is prepared by the clinic and the interpretation of laboratory tests.

Fig. 69.8 Renal compensatory mechanism in metabolic acidosis. Na,HPO, phosphate pathway.


EXTRACELLULAR
SPACE
TUBULAR
CELL
TUBULE
LIGHT
H,C0,
Na,HPO,
HCO,
H+
na
NaHPO,
NaH,P0,
NaHCO,
NaHCO,

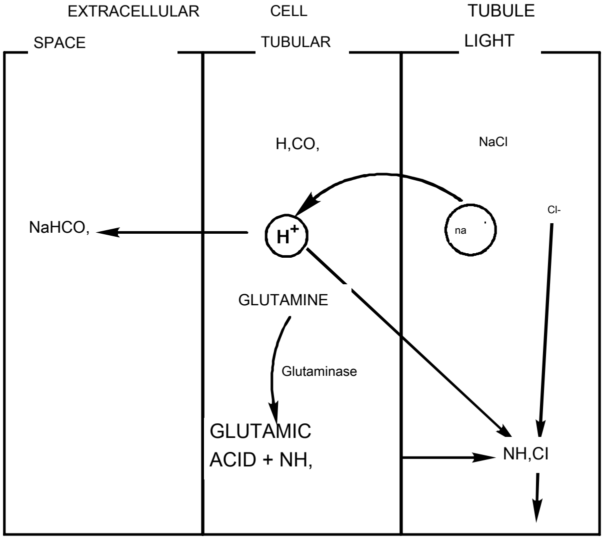

EXTRACELLULAR
SPACE
CELL
TUBULAR
TUBULE
LIGHT
H,CO,
NaCl
NaHCO,
H+
na
Cl-
GLUTAMINE
Glutaminase
GLUTAMIC
ACID + NH,
NH,Cl

Fig. 69.9 Renal compensatory mechanism in metabolic acidosis. Ammonia pathway (NH,).

Clinical picture

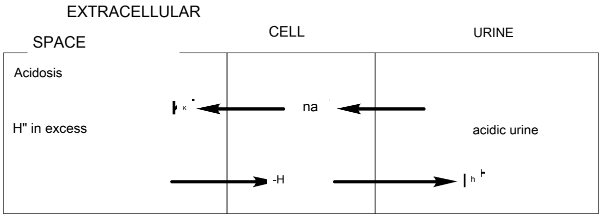
In addition to the symptoms and signs of the causative condition, for example, the ketonic breath of decompensated diabetes or the urinary breath of end-stage renal failure, a patient with decompensated acidosis will present with deep breathing (air hunger), which can lead to Kussmaul type breathing, headache, stuporous state, hyporeflexia, paresthesias and great asthenia, as well as serious alterations of consciousness that can reach comma. Digestive symptoms such as sudden anorexia, nausea, vomiting, diarrhea and intestinal paresis may occur. It is worth noting abdominal pains that can simulate an acute abdomen.

Complementary exams

There is low alkaline reserve, high [H] and pH below 7.35 (in decompensated ones).

The urine is acidic and the pCO is decreased. Potassium is increased (in acidosis there is hyperkalemia, as we can see in the diagram in Figure 69.10.

Fig. 69.10 Mechanism of hyperkalemia in metabolic acidosis.


EXTRACELLULAR
SPACE
CELL
URINE
Acidosis
H" in excess
na
acidic urine
-H

Etiology

Metabolic acidosis is classified according to whether it has normal or elevated remaining anions (RA); with normal remaining anions, depending on whether the serum K* is normal or low or the serum K* is high; and with high remaining anions, depending on whether the osmolal gap is normal or high.

The causes of metabolic acidosis, according to this classification, are shown in Table 69.5.

In pure metabolic acidosis with normal remaining anions, as much as bicarbonate is reduced, chlorine is increased, that is, the changes produced (A) in these two anions are quantitatively equal and therefore, the remaining anions are not modified.

A HCO, = A CI

However, in pure metabolic acidosis with elevated remaining anions, as bicarbonate is reduced, remaining anions are elevated.

HCO, = AAR

If the elevation of the remaining anions is greater than the decrease in HC0,-, there is an added metabolic alkalosis.

Respiratory acidosis

Concept

In these cases there is a tendency to decrease the pH of the blood due to a primary decrease in the index

Table 695 Etiology of metabolic acidosis with normal or elevated remaining anions and their respiratory compensation

Causes of metabolic acidosis			
With remaining anions normal is		With remaining anions the evades	
[K] Normal or low	[K] Al to	oSmol gap to normal	Evada osmol gap
- Proximal tubular acidosis (TA)	- Initial kidney failure	- Starvation	- Toxic:
- Distal AT (type I)	- Hydronephrosis	Diabetes mellitus -	Methanol
Buffer deficiency:	- Acid administration:	Alcoholism	Ethylene Glycol
Phosphates	ammonium chloride	Lactic acidosis	
Ammonium	Arginine Lysine	- Toxic: Salicylates	
Diarrhea	- Hypoaldosteronism -	- Terminal chronic renal	
Pancreatic fistulas	Distal AT type IV	failure	
Posthypocapnia	(hyperkalemic)		
- Urinary diversions:	- Primary hyperparathyroidism		
Ureterosigmoidostomy Ureter-ileal Bladder-ileal			
Respiratory compensation			
PCO, 1.5 (HC0,) + 8 t 2 PCO, >: Mixed acidosis defect metabolic + respiratory acidosis PCO, <: Mixed defect metabolic acidosis + respiratory alkalosis			
Pure metabolic acidosis with normal remaining anions ACH= A HCO, (Hyperchloremia) (metabolic acidosis + metabolic alkalosis)		Pure metabolic acidosis with high remaining anions A AR = A HCO, Yeah A AR < A HCO,	

of pulmonary ventilation, with an increase in the partial pressure of CO, which determines an accumulation of carbonic acid in the blood. It can have an acute or chronic evolution.

Syndromogenesis or physiopathology

All diseases capable of producing respiratory acidosis have a common denominator: reduction in pulmonary ventilation, which inevitably leads to tissue hypoxia.

The primary alteration of alveolar ventilation with CO retention leads to an acid-base imbalance, and in response to this the body puts into action the buffer mechanism and other compensatory mechanisms, to avoid a decrease in the bicarbonate/carbonic acid ratio that lowers the pH of the blood.

In this process, there is also an increase in the excretion of acid metabolites through the urine.

Syndromography or positive diagnosis

Clinical picture

It is given by the symptoms and signs of the underlying disease.

In cases of narcosis or CO poisoning, respiratory rhythm disorders, muscle weakness, irritability, disorientation and stupor that can lead to coma occur. Sometimes breathing is accelerated, in an attempt by the respiratory center to eliminate CO2.

Complementary exams

There is a high alkaline reserve (this information seems paradoxical, which is why this test is not very useful to express alterations in the acid-base balance); the pH is low (in the decompensated ones); the urine is acidic; there is polyglobulia in chronic cases; pCO is elevated.

Etiology

1. CNS Depression:
   - a) Sedatives.
   - b) Trauma.
   - c) Ischemia.
2. Neuromuscular diseases:
   - a) Myopathies.
   - b) Severe K depletion.
   - c) Guillain Barré disease.
3. Limitations of the chest wall:
   - a) Kyphoscoliosis.
   - b) Scleroderma.
   - c) Trauma.
4. Difficulty in respiratory mechanics:
   - a) Pleural effusion.
   - b) Pneumothorax.
5. Acute lung diseases:
   - a) Aspiration.
   - b) Spasm.
   - c) Tumor.
6. Chronic obstructive pulmonary diseases.
7. Severe pneumonia.
8. Cardiopulmonary arrest.

alkalosis syndrome

It is a physiological disorder that tends to produce an increase in the pH of the blood, which will occur if compensatory phenomena do not intervene.

Alkalosis will exist whenever there is an excess of bases in the blood or a deficiency of acids.

There are two types of alkalosis: metabolic and respiratory. In the first there is a primary metabolic disturbance and in the second there is a primary respiratory

metabolic alkalosis

Concept

It is characterized by the gain of a strong base or the loss of a strong acid and a negative balance of hydrogen ions [H*] occurs.

Syndromogenesis or pathophysiology

Excess bases can be produced by excessive ingestion of bicarbonate or by loss of acid ions, as in cases of vomiting or gastric aspiration.

The loss of hydrogen ions may also be due to a depletion of K*, since in these cases intracellular K* is exchanged for extracellular H, as we have already seen. This explains why there is alkalosis in hypokalemia.

As a compensation mechanism, there is a decrease in pulmonary ventilation to retain CO, and an elimination of basic elements through the urine.

Buffers and other compensatory mechanisms come into action to prevent an increase in pH.

Syndromography or positive diagnosis

Clinical picture

Breathing is shallow, there are tendon jumps, irritability and in severe cases tetany occurs.

Complementary exams

There is increased alkaline reserve, pH above 7.45 (in decompensated ones), CI and K* are decreased and Nat is normal or low.

Etiology

metabolic alkalosis

Metabolic alkalosis is due to three groups of causes:

1. With volume contraction and arterial hypotension.
2. With volume expansion and increased blood pressure,
3. With alkali overload.

According to these groups and the different mechanisms involved, the etiology can be:

A. Volume contraction and arterial hypotension.

1. Gastrointestinal losses:
    - a) Vomiting.
    - b) Gastric aspiration.
    - c) Gastric fistula.
    - d) Congenital chlororrhea.
    - e) Villous adenoma.
2. Renal loss Na, CI, K, H:
    - a) Diuretics.
    - b) VPRE (effective renal plasma volume).
    - c) Heart failure, liver cirrhosis, nephrotic syndrome.
    - d) Non-resorbable anions.
    - e) Penicillins.
    - f) Hypercalcemia.
    - g) Mg.
    - h) PTH (parathyroid hormone).
    - i) Posthypercapnia.
    -) Bartters syndrome.

B. Volume expansion and elevated blood pressure.

1. Decreased plasma renin:
    - a) Primary aldosteronism.
    - b) Enzymatic defects (11ß and 17 - hydroxysteroids)
    - c) Cushing syndrome.
    - d) Administration of cortisone.
2. Increased plasma renin:
    - a) Kidney tumor.
    - b) Severe arterial hypertension.
    - c) Renal artery stenosis.
    - d) Treatment with estrogens.

C. Alkali charge.

1. NaHCO,.
2. Lactate-acetate.
3. Citrate.

Metabolic alkalosis with volume expansion can in turn be divided into hyporeninemic and hyperreninemic.

For clinical differentiation between metabolic alkalosis with volume contraction and volume expansion, the concentration of chloride in the urine is used, as seen in Table 69.6.

Respiratory alkalosis

Concept

It is an abnormal physiological process in which the degree of alveolar ventilation is increased in relation to the production of CO, which determines a deficit of H,CO, in the blood. It is also called hypocapnia.

Table 626 Etiology of metabolic alkalosis with volume contraction and expansion

Acid-base balance Causes of metabolic alcal osİs	
Chlorine sensitive (CH in urine < 20 mE g/L)	Chlorine resistant (IC- in urine > 20 mEq/L)
Vomiting	Hyperaldosteronism
Nasogastric suction	Cushing syndrome
Congenital chlororrhea	Administration of corticosteroids
Villous adenoma	Bartter syndrome
Diuretics	Severe potassium depletion

Syndromogenesis or pathophysiology

This disorder can be produced by different mechanisms, depending on the cause that causes it.

Stimulation of the respiratory center is the most frequent mechanism; It can be caused directly by organic lesions of the central nervous system or indirectly by stimulation of peripheral chemoreceptors (in hypoxemia) or by stimulation of the Hering-Breuer reflex in localized respiratory diseases (pneumonia).

Any of the aforementioned mechanisms leads to an excessive loss of CO, which causes a decrease in H,C0, in the blood, therefore leaving free the Na that produces alkalosis.

In response to this alteration of the acid-base balance, two mechanisms act secondarily that tend to reduce the disturbance caused:

1. Buffer mechanism.
2. Compensating mechanisms.

Through them, the excretion of phosphate and bicarbonate by the kidney is reduced and the proportional excess of bases is eliminated, until the ratio of free CO/combined CO returns to normal.

Syndromography or positive diagnosis

Clinical picture

It is similar to metabolic alkalosis; hyperpnea, muscle hypertonia and hyperreflexia, and tetany predominate in advanced cases.

The neuromuscular excitability of alkalosis is determined because in the characteristic humoral relationship of:

$$\frac{NaK+OH}{Ca*Mg+ *H+}$$

There is a decrease in hydrogen ions as a consequence of alkalosis, since this ratio of ions is the that maintains the balance of neuromuscular excitability or inhibition.

Complementary exams

There is normal or low alkaline reserve, high pH (in decompensated ones). Urine is alkaline. There may be hypokalemia and the pCO2 is low.

Etiology

1. By direct stimulation of the respiratory center, as seen in meningoencephalitis, hysteria, salicylate poisoning and hyperthermia.
2. During hypoxemia (high altitudes).
3. In localized respiratory diseases (pneumonia).
4. In hyperventilation syndrome, if it is maintained:
   - a) Anxiety.
   - b) CNS conditions:
     - Infection.
     - Tumor.
   - c) Drugs and hormones:
     - Salicylates.
     - Catecholamines.
     - Progesterone.
     - Analeptic overdose.
   - d) Fever.
   - and) Sepsis due to gram-negative germ.
   - F) Endotoxins.
   - g) Hyperthyroidism.
   - h) Hypoxia.
   - i) Mechanical respirator.
   - i) Pregnancy.
   - k) Liver failure.
   - 1) Mild pulmonary edema.
   - m) Lung diseases.

SEMIOLOGY OF THE MALE AND FEMALE REPRODUCTIVE SYSTEMS

70

MALE REPRODUCTIVE SYSTEM

The anatomy, physiology and physical examination techniques of the male reproductive system were already discussed in volume 1, Chapter 19. Here we will briefly point out some important data that we must keep in mind during the interview, the most frequent pathological processes and the alterations of the genital organs detected by the physical examination.

INTERROGATION 0 Anamnesis

During the interrogation we must collect the causes and evolutionary course of the condition that motivates the consultation, but given the nature of some points (sexual emotional life, anomalies and deviations, etc.), which are sometimes hidden due to modesty, the doctor must display all his skill and carry out the interrogation naturally and carefully, in order to obtain true answers.

MOST FREQUENT PATHOLOGICAL PROCESSES

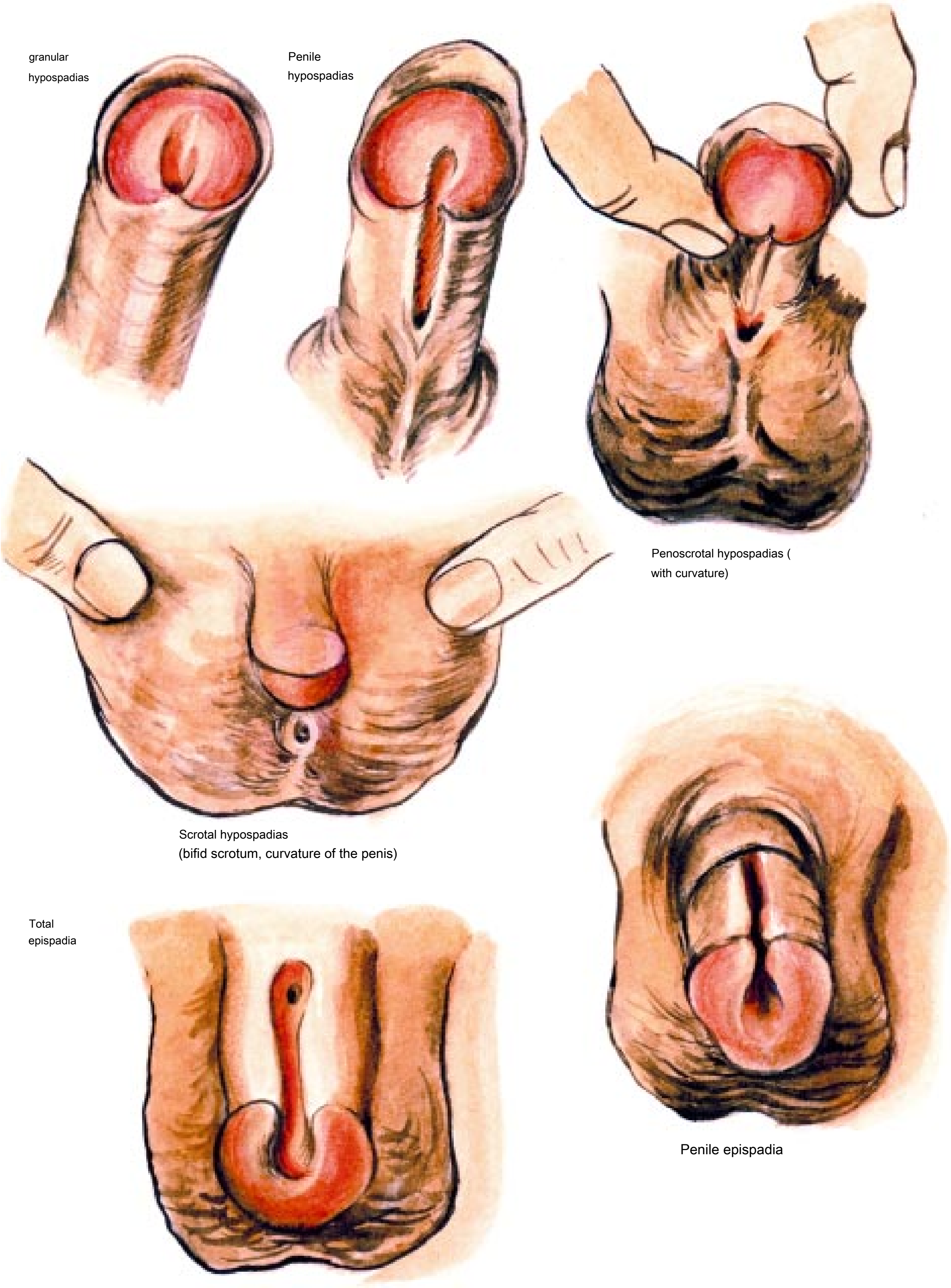
The patient's disorders may be congenital, such as epispadias, hypospadias (Fig. 70.1) and phimosis (Fig. 70.2), or acquired, in the course of the following processes:

Infectious. Syphilis, gonorrhea, urethritis due to trichomonas or monilias, chancroid, lymphogranuloma venereum, among others.

Nervous. Coeundi impotence associated with bladder and rectal disorders, in neurological lesions of the sacral cord or cauda equina (remember that the erector and ejaculatory centers are very close to the bladder and rectal centers); other times this occurs in tabes (spinal cord lesion of the tertiary period of syphilis), in plaque sclerosis, etc.; However, in most cases, impotence is related to psychological alterations.

Endocrine. In pituitary, thyroid and adrenal insufficiencies it is common for there to be alterations in the performance of intercourse.

Toxins and medications. Alcohol, cocaine, morphine, ganglioplegics (hexamethonium, guanethidine) and drugs act as depressants.



granular hypospadias

Penile hypospadias

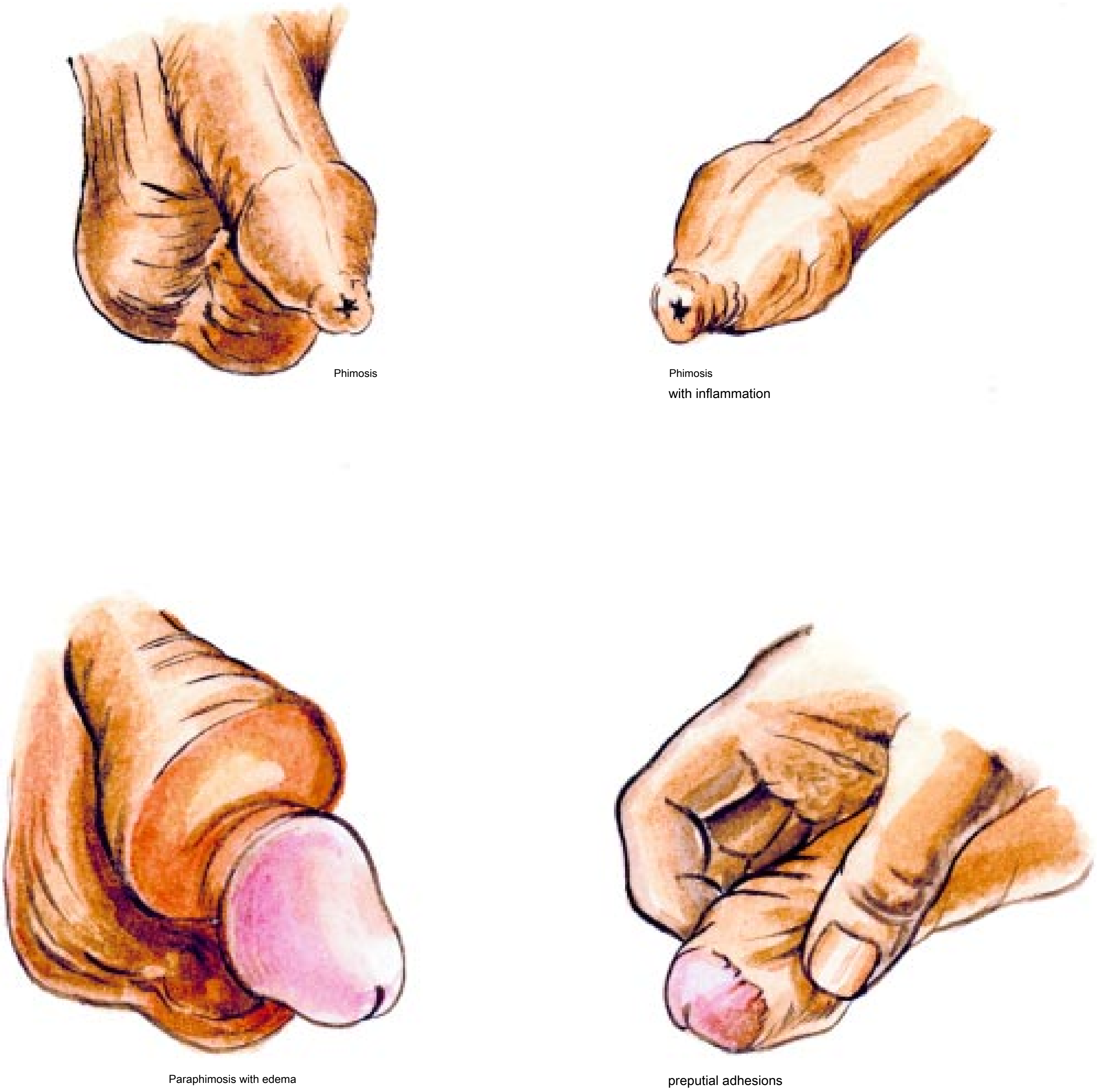
Penoscrotal hypospadias (with curvature)

Scrotal hypospadias (bifid scrotum, curvature of the penis)

Total epispadia

Penile epispadia

Fig. 70.1 Epispadia and hypospadia.



Phimosis

Phimosis
with inflammation

Paraphimosis with edema

preputial adhesions

Fig. 70.2 Phimosis, paraphimosis and preputial adhesions.

psychotropic drugs (barbiturates, bromides). Yohimbine, iproniazid, and others act as stimulants (aphrodisiac drugs).

DETECTABLE ALTERATIONS ON THE PHYSICAL EXAMINATION

Inspection

You should begin with the general examination followed by the local examination, which includes the visual examination of the penis, scrotum, inguinal regions and perineum, as explained in volume 1, Chapter 19. Alterations in height, fat distribution, primary and secondary sexual characteristics were mentioned in said volume and will be studied in more detail in the chapters dedicated to the endocrine system.

From the local point of view, in the penis they can contrast:

Congenital morphological alterations, already mentioned, such as epispadias, hypospadias (deformities in which the urethra opens on the dorsum and the underside of the penis, respectively) (see fig. 70.1) and phimosis, which consists of the narrowness of the opening of the foreskin, resulting in the impossibility of discovering the glans (see fig. 70.2).

Ucerations, of which the most frequent are venereal, which almost always sit on the mucosa of the glans, at its base next to the frenulum, or in the balano-preputial groove. They generally correspond to syphilitic chancres (fig. 70.3), soft chancres (fig. 70.4), multiple ulcerations due to trichomonas or monilia, with or without secondary infection, etc. Sometimes it even leads to gangrene. Parasitic diseases caused by Pediculus pubis or scabies are very common (fig. 70.5).

Less frequently seen is cancer of the penis, which constitutes 1% of all cancers in men (Fig. 70.6).

Continuing with the local inspection, we will also observe the scrotal bags (which contain the testicles, epididymides, vessels, etc.), where we will frequently find an increase in size due to hydrocele (accumulation of fluid in the vaginal cavity) (fig. 70.7);
varicoceleo varicose dilation of the spermatic veins, which can be unilateral or bilateral (accompanied by sterility in many cases) (fig. 70.8); inguinal-crotal hernias; inflammatory or neoplastic tumors of the testicles, etc.

Palpation

Palpation of the scrotum

When the scrotum is palpated, in the event that there is edema (due to local or neighboring inflammations, circulatory or renal failure, alterations in protein plasma, venous or lymphatic obstructions, etc.), the skin offers a pasty resistance and leaves a mark (fovea) to pressure; In emphysema (pneumoscrotum) it is easy to recognize the flaccidity and elasticity of the integuments, as well as a slight crepitation (as if small dry and friable vesicles were crushed); The aricocele awakens a special sensation compared to that caused by a bunch of worms or chicken guts; benign tumors (for example, sebaceous cysts), malignant ones, syphilitic chancre, and others, stand out well for their hardness.

Palpation of the tunica serosa vaginalis

The vaginal serosa impingement sign, described in Chapter 19 of Section I, volume 1, helps establish the differential diagnosis between testicular tumors and pachyvaginalitis of the vaginal serosa or hematocele.

Palpation of the testicle and epididymis

It is checked whether the testicles occupy their normal place or are ectopic, and demonstrate if there is any alteration in the size, surface, consistency and/or pain caused, both in any testicle and in each epididymis.

Increased tannin of the testicle

It is easily verifiable by palpation. It is caused by different injuries.

Semiogenesis and semidiagnosis

1. Increased fluid in the testicular vaginal cavity: hydrocele (see Fig. 70.7).
2. Dilation of the spermatic veins: varicocele (see Fig. 70.8).
3. Acute venous congestion due to torsion of the spermatic cord: testicular torsion (Fig. 70.9).
4. Inflammatory swelling: orchitis, epididymitis and epididymitis (Fig. 70.10), testicular hematoma.
5. Tumor enlargement: malignant tumor of the testicle (seminoma, teratoma, choriocarcinoma).

Palpation of the vas deferens and spermatic cord

The vas deferens increases in thickness and sensitivity in acute tuberculous, gonorrhagic and non-specific processes. In tuberculosis it thickens unevenly, taking on a rosary appearance. It appears unscathed in syphilis and in malignant and benign tumors of the testicle; In seminomas, the spermatic artery is sometimes perceived to be excessively pulsating, an index of the hypervascularization of this hormonal tumor.

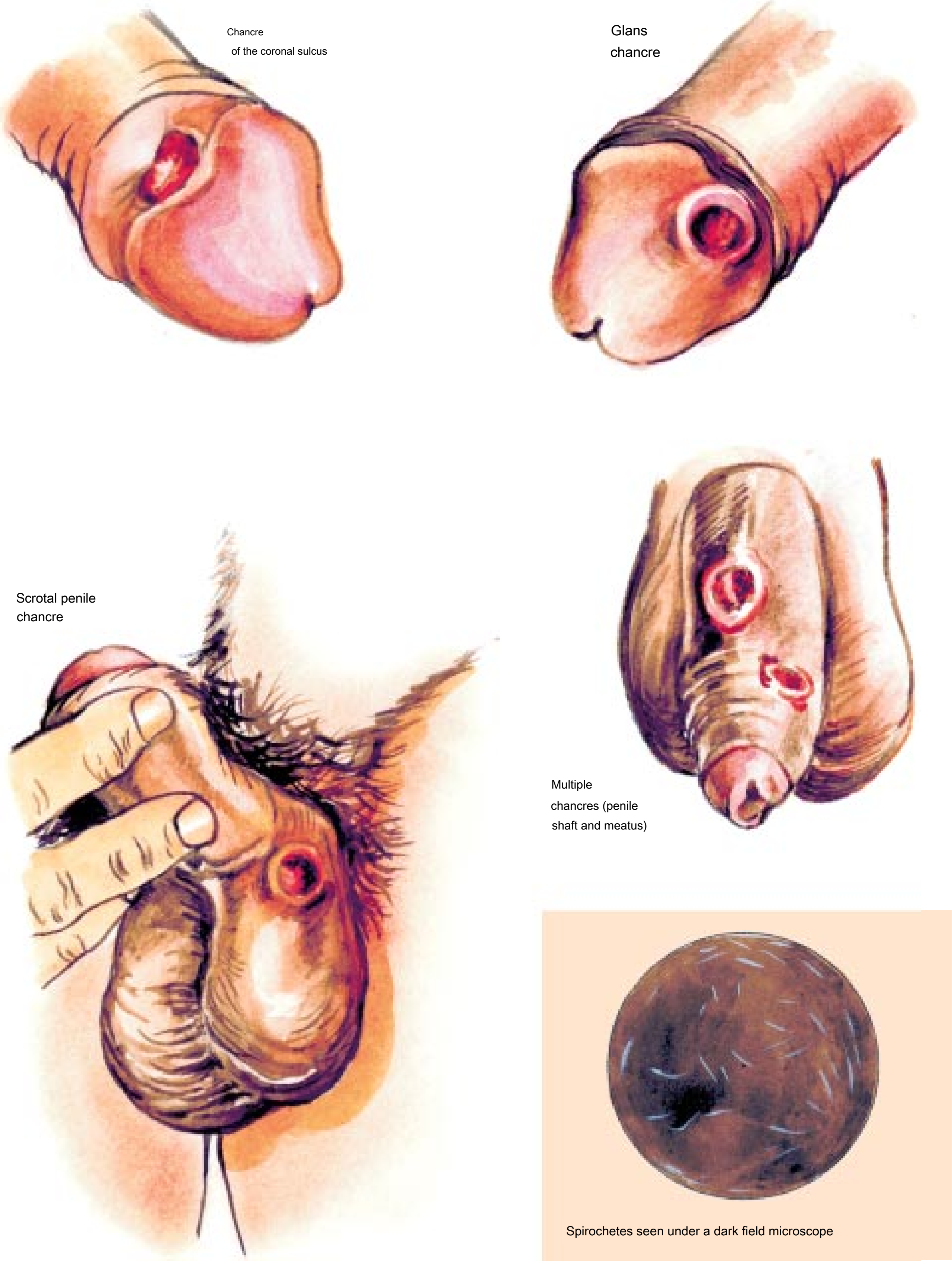

Chancre
of the coronal sulcus
Glans
chancre
Scrotal penile
chancre
Multiple
chancres (penile
shaft and meatus)
Spirochetes seen under a dark field microscope

Fig. 70.3 Syphilitic chancre.

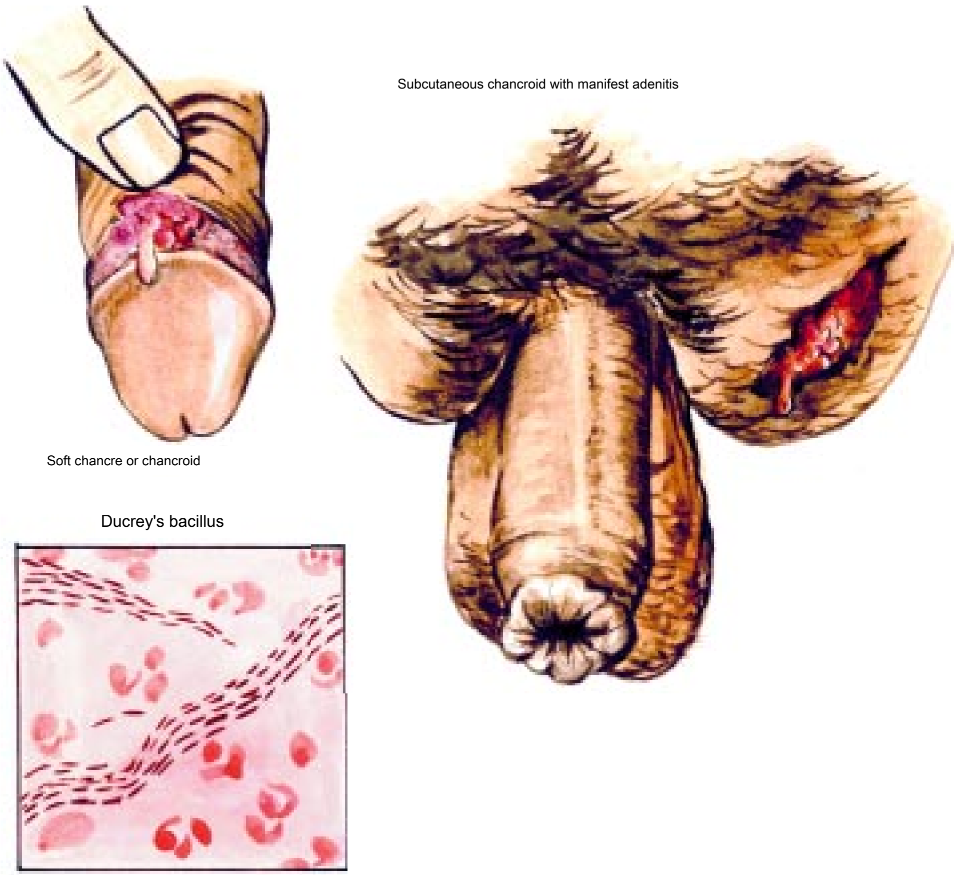

Subcutaneous chancroid with manifest adenitis
Soft chancre or chancroid
Ducrey's bacillus

Fig. 70.4 Soft chancre.

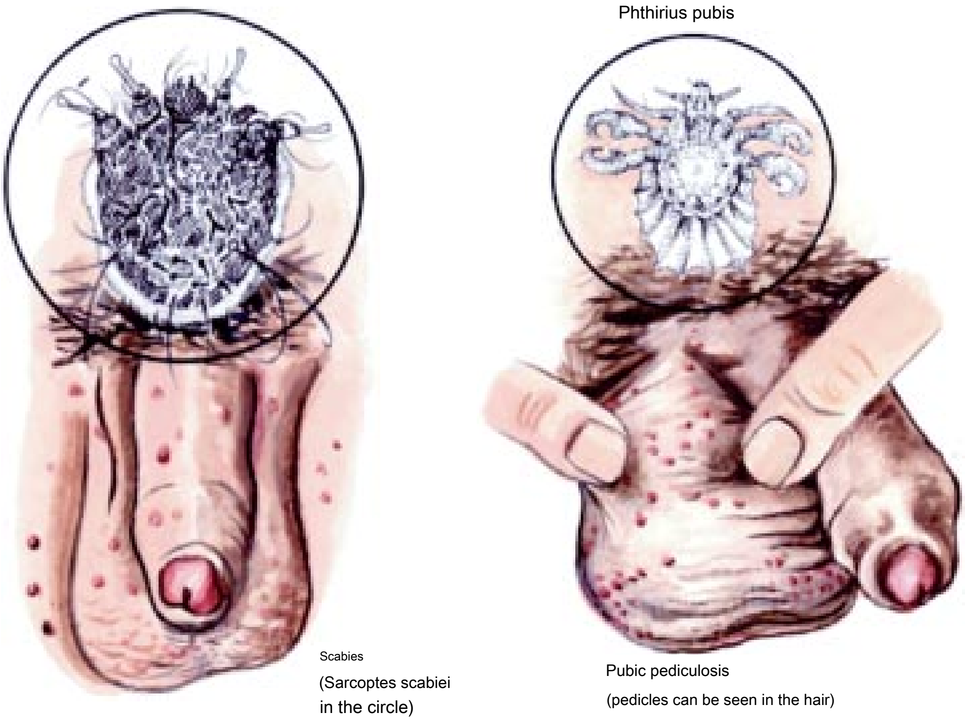

Phthirius pubis
Scabies
(Sarcoptes scabiei
in the circle)
Pubic pediculosis
(pedicles can be seen in the hair)

Fig. 70.5 Scabies and pediculosis pubis.

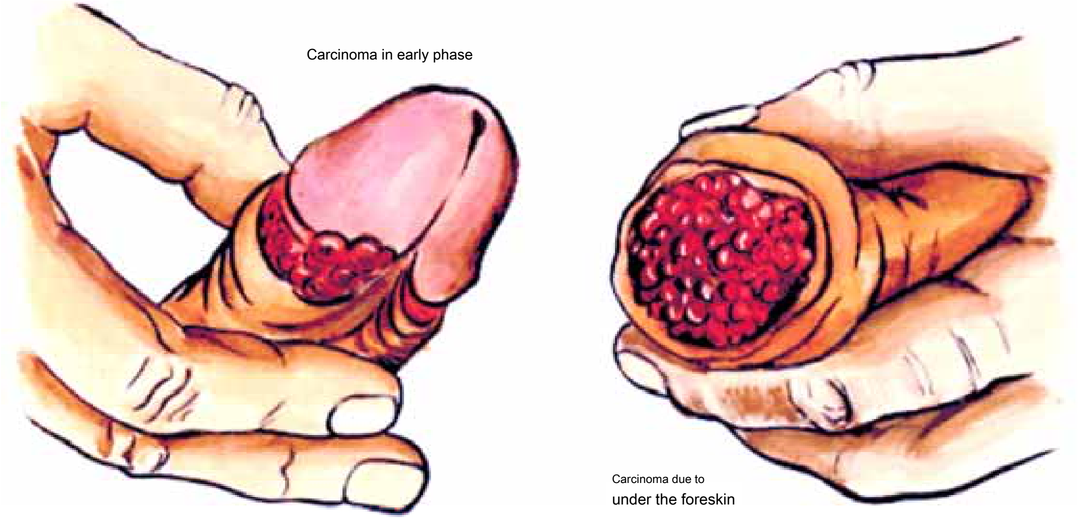

Carcinoma in early phase
Carcinoma due to
under the foreskin

Fig. 70.6 Cancer of the penis.

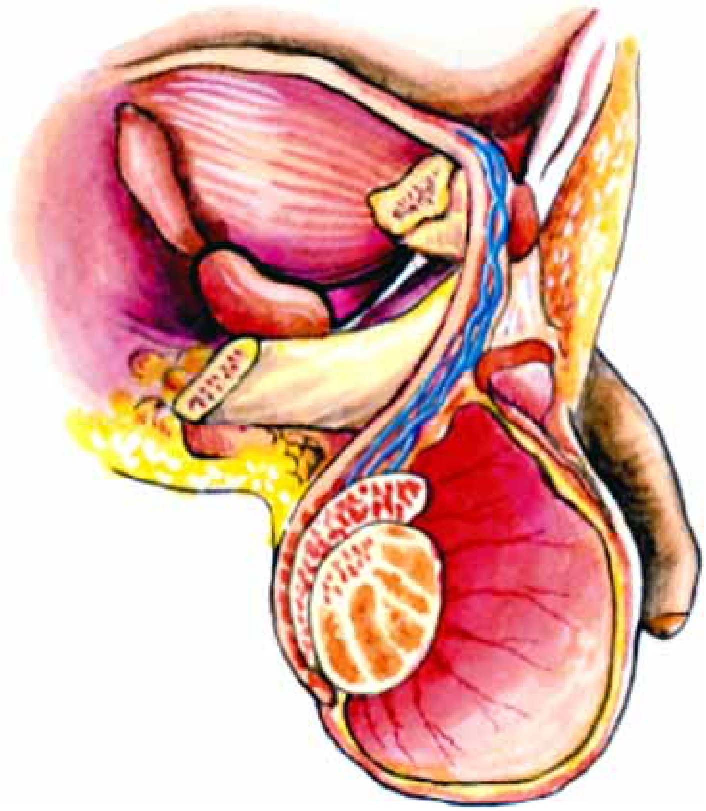
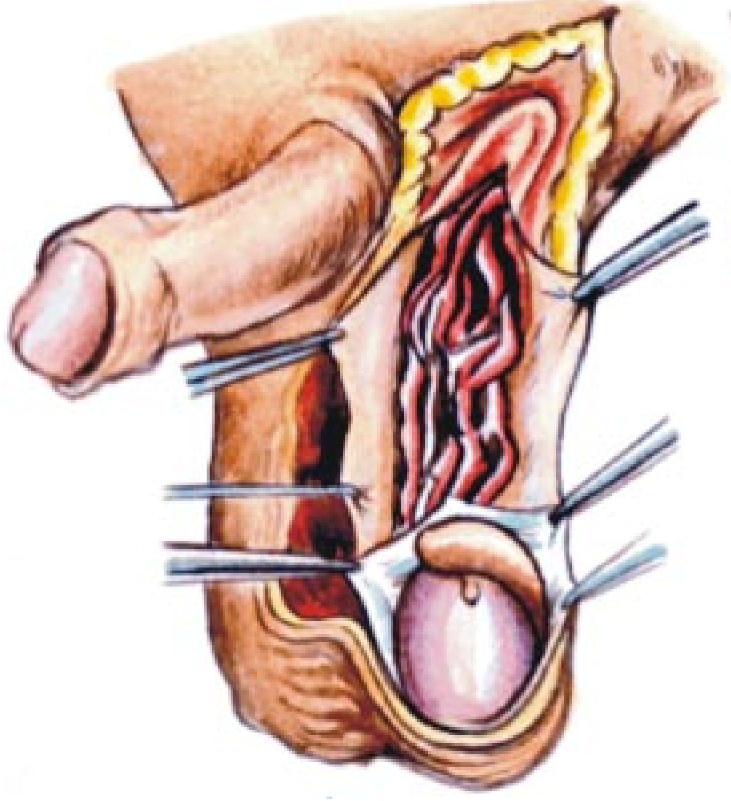


Fig. 70.7 Simple hydrocele.



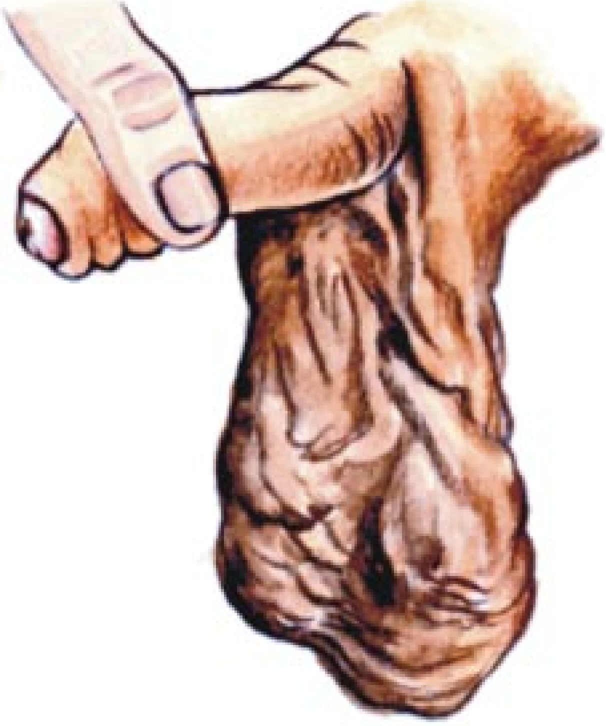
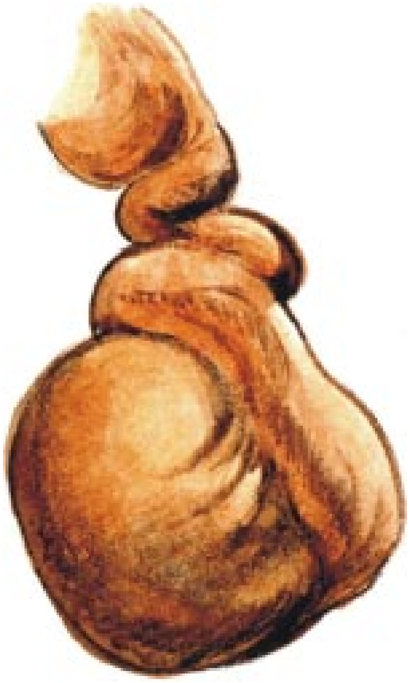
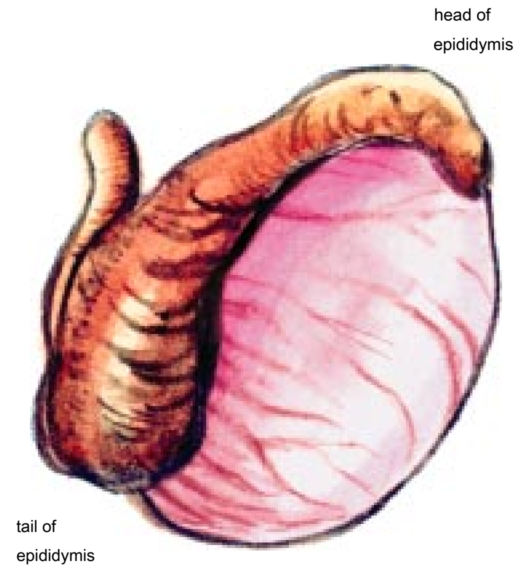


Fig. 70.8 Varicocele.

Fig. 70.9 Testicular torsion.

Fig. 70.10 Acute epididymitis orchitis.




head of epididymis
tail of epididymis

Cysts and tumors of the spermatic cord have been reported; Malignant ones (20% of cases) manifest as a painful inguinal tumor, accompanied by an inflammatory reaction of the skin of the scrotum and the base of the penis.

Prostate enlargement

In these cases, its size can vary between 4 and 8-10 cm or more, according to its etiology.

Semiogenesis and semidiagnosis

1. Benign hyperplasia of the gland: prostatic adenoma (fig. 70.11).
2. Malignant tumor of the gland: prostatic adenocarcinoma (fig. 70.12).
3. Inflammatory swelling: prostatitis and prostate abscess (fig. 70.13).

Palpation of the urethra

Soft or hard infiltration plaques may be perceived in urethritis and periurethritis, as well as hard areas in relation to a syphilitic chancre or small glandular abscess (both in the first centimeters of the duct) or foreign bodies. Palpation with the help of the Beniqué probe can recognize the existence of small nodules in the inflammation of the Littré glands (litritis).

FEMALE REPRODUCTIVE SYSTEM

It is recommended to first review the anatomy, physiology and physical examination techniques of the female reproductive system, in volume 1, Section I, Chapter 20.

The gynecological clinical history, like any specialized clinical history, must be aimed at clarifying the condition of the consultant, emphasizing, fundamentally, everything related to the genital apparatus and its annexes.

INTERROGATION 0 Anamnesis

The reason for consultation must very specifically reflect the fundamental reason for your visit and the history of the current illness (HEA) will contain all the data necessary for the diagnosis.

In every gynecology consultation there must be a printed form where, in a logical sequence, in addition to the reason for consultation and the AH, the patient's gynecological and obstetric history is recorded, such as: age at menarche; menstrual formula; date of last menstruation; history of pain, colporrhea and menstrual disorders; menopause and climacteric disorders; wildebeest-group of births, abortions, as well as everything related to them; operations; family and personal history of interest; etc These same data must also be collected in the clinical record of every patient admitted for any reason.

MOST FREQUENT GYNECOLOGICAL SYMPTOMS

Pain

It is a common symptom, for which many women go to the gynecological consultation and it can be the dominant symptom or the one that bothers the patient the most. The pain that accompanies menstruation, called dysmenorrhea, must be differentiated. The pain referred by women to the genital tract is usually located in the hypogastrium (uterine pain) or in the iliac or lumbar fossae (ovarian pain) and will have very varied manifestations in frequency, intensity, duration and rhythm, according to the condition that causes it and its evolution.

Less frequently, pain is referred to the external genitalia.

Semiogenesis or pathophysiology

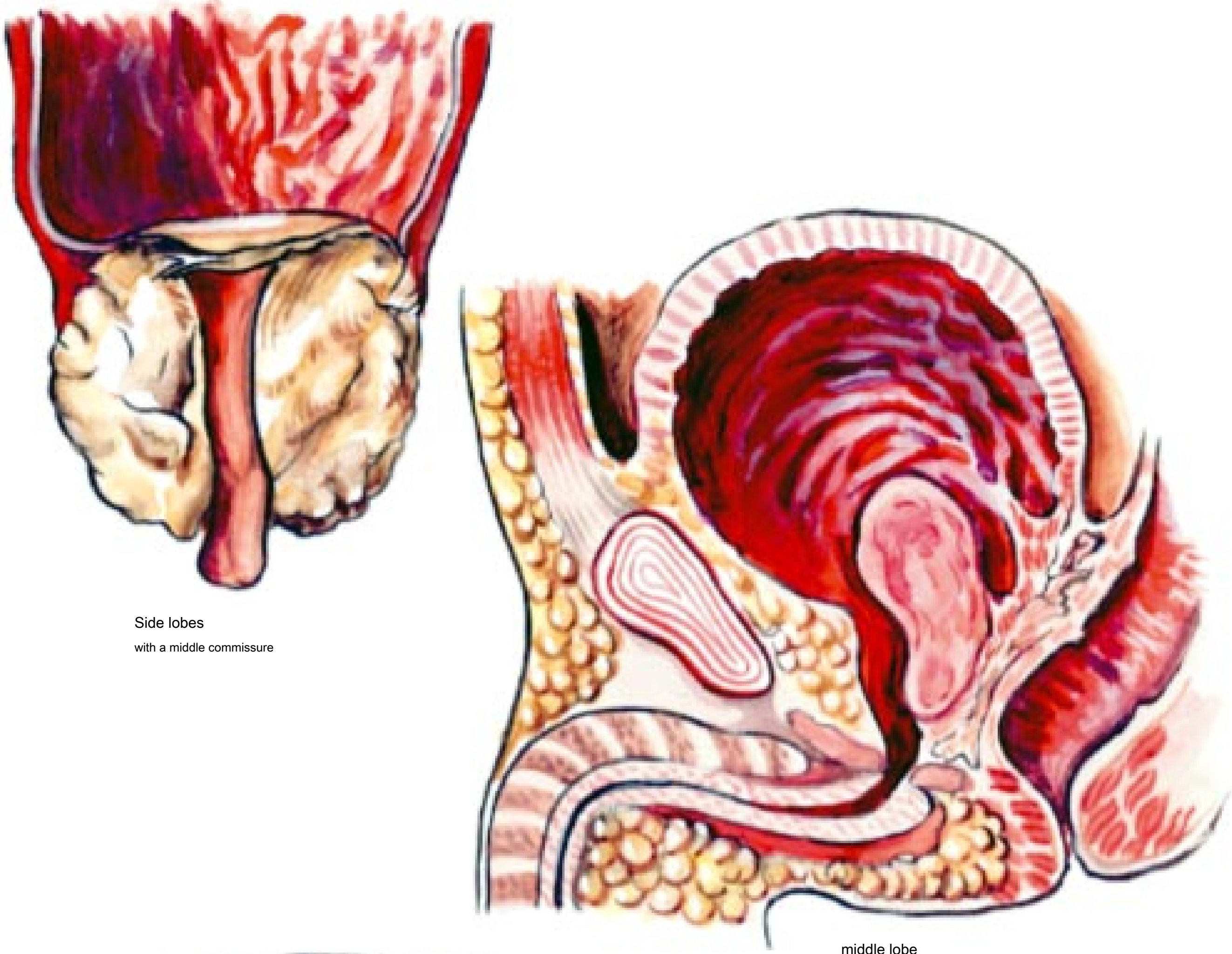
Directly located pain occurs in the lower third of the vagina and in the vulva, due to having sensory innervation from the hypogastric plexus and the internal pudendal nerve, as observed in bartholinitis, trauma, vulvitis, etc.

Indirect pain is produced by stimulation of the pelvic peritoneum that surrounds the internal genital organs. It originates in the ovaries, tubes, uterus, and upper portion of the vagina; It is transmitted neurovegetatively and is referred by a viscerocutaneous reflex to the corresponding metameric area, through the posterior horns of the spinal cord.

Semiotechnics

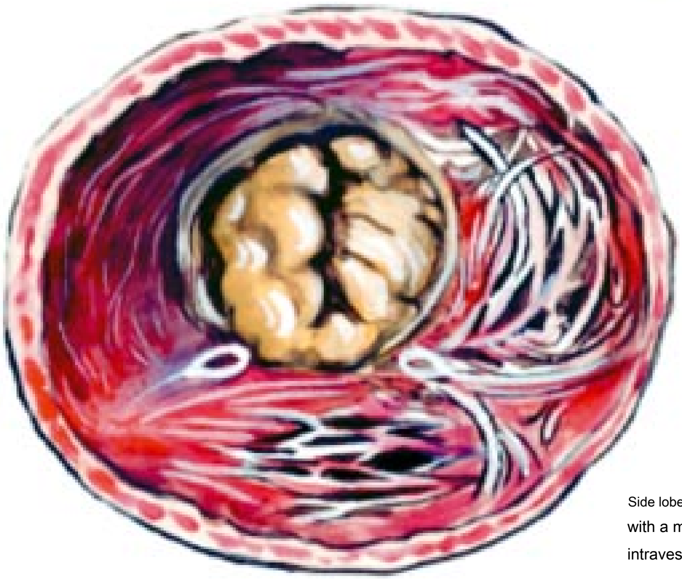
By inspection and palpation of the abdomen, the location of the pain, its radiation and extension, as well as the existence or not of peritoneal irritation, can be determined.

The vaginal examination will allow us to better specify the pain caused at the level of the different parts of the genital system: cervix, uterine body, tubes, ovaries, parametria, as well as the increase in volume in these structures, their consistency, shape, size and location. Through this examination we can also appreciate the characteristics of the cul-de-sac (especially the posterior one), which can be painful and reluctant or pasted, depending on the process that the pain causes.



Side lobes
with a middle commissure

middle lobe



Side lobes
with a middle commissure (
intravesical aspect)

Fig. 70.11 Prostatic adenoma.

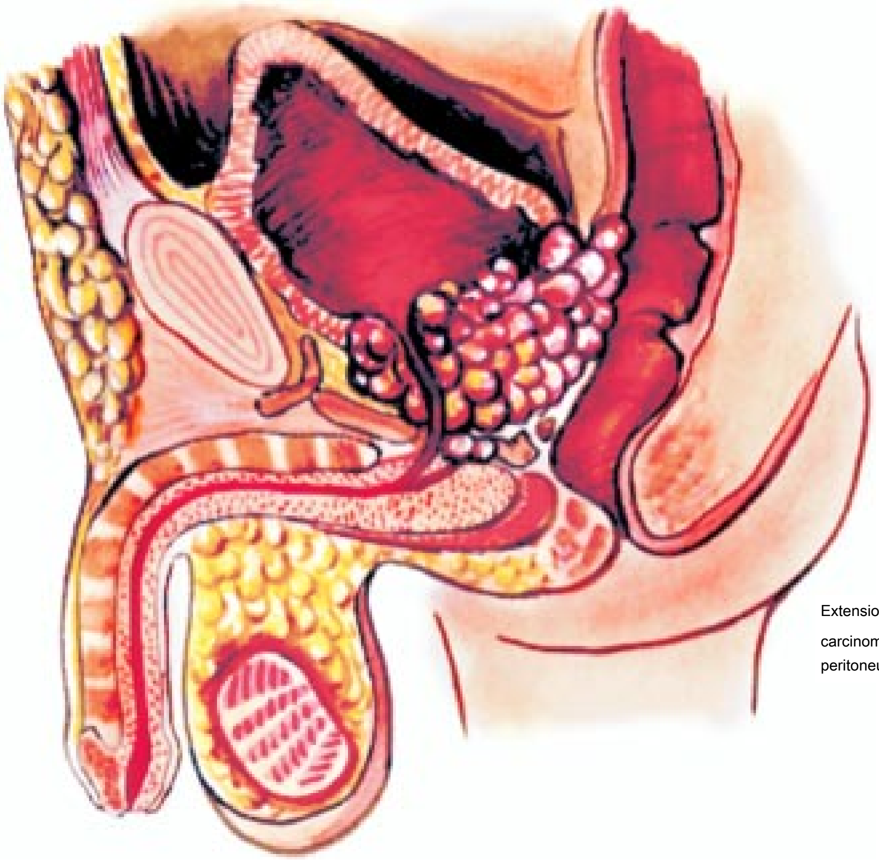


Fig. 70.12 Prostatic adenocarcinoma.

Extension of prostate carcinoma to the bladder, peritoneum and rectal wall

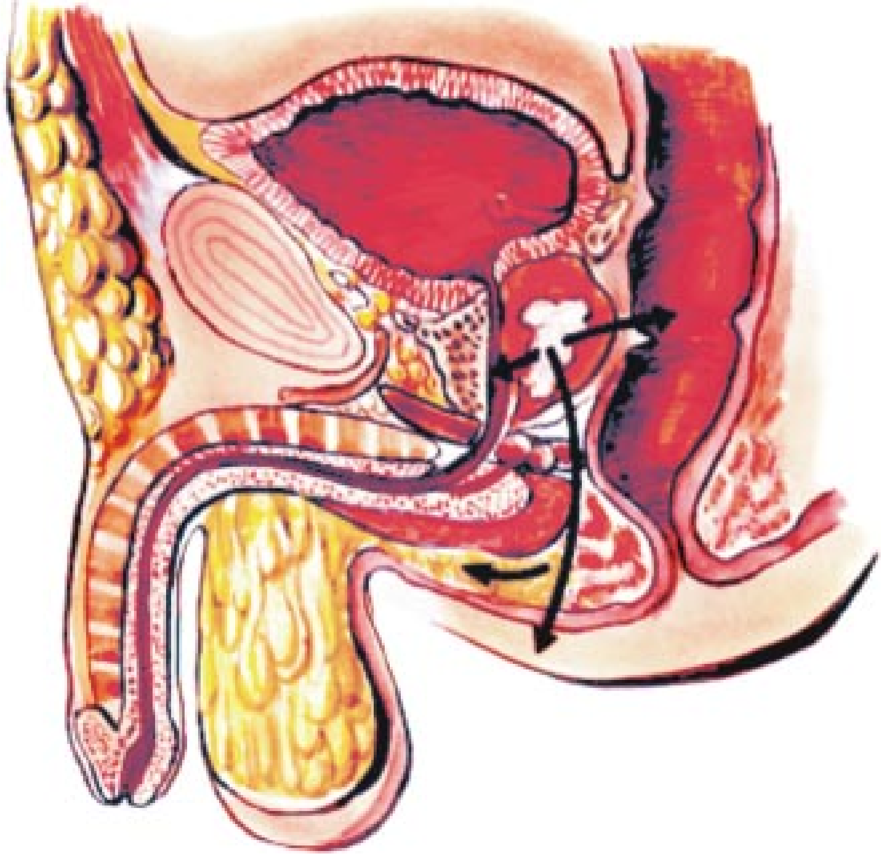


Fig. 70.13 Prostatic abscess.

Semiography

1. Acute pain:

a) Ovulation pain: its rhythm of presentation in the middle of the cycle, 14-15 days, without other accompanying symptoms and its progressive relief, as well as its frequent repetition in the same woman, allows it to be differentiated from other pains, which makes it considered in the clinic as a very reliable sign of ovulation.

This allows sexual relations to be planned according to the woman's interests and her desire to have children or not. This pain is caused by the rupture of the follicle and the small intraperitoneal hemorrhage that it causes.

b) Pelvic pain in acute pelvic inflammation is accompanied by fever, which can be high (39-40°C), with general malaise, chills, tachycardia and leukorrhea, sometimes purulent and sometimes foul-smelling. In these cases there is frequently a history of childbirth, abortion, electrocoagulation of the neck, insufflation, hysterosalpingography, placement of an intrauterine device or intercourse.

c) In a ruptured or fissured tubal pregnancy, there is a history of possible pregnancy symptoms; The pain is frequently more diffuse, sometimes reflected in the shoulders due to phrenic irritation, and on physical examination an adnexal tumor can sometimes be palpated.

d) In the complications of ovarian cysts, there may be a previous diagnosis of the cyst or its discovery in the gynecological examination.

e) In primary or functional dysmenorrhea, pain, as the predominant symptom, is colic, located in the hypogastrium with lumbosacral irradiation; other times it is a sustained hypogastric pain. It may be accompanied by digestive symptoms (nausea and vomiting), nervous disorders (headache, dizziness, excitability and mental instability, depression) and manifestations of premenstrual tension (mastalgia, edema, weight gain, sensation of abdominal fullness) .

f) In dysmenorrhea secondary to other conditions, or symptomatic, there is no symptomatic courtship referred to and the physical examination may allow the diagnosis of: uterine hypoplasia, pelvic endometriosis, chronic pelvic inflammation, fibroids, large endocervical polyps, cervical stricture, uterine retroflexion or pelvic congestion.

2. Chronic pain:

a) Pelvic endometriosis produces dysmenorrhea that appears late in a woman's life (30 years or more), which progressively increases in intensity and is frequently associated with sterility. On physical examination, multiple painful nodules may be found; irregularities in the uterus, in the lateral and posterior fornix, and in the ovaries.

In long-standing cases, endometriotic nodules can reach 15-20 cm in diameter and create very firm and painful adhesions to the bladder, rectosigmoid, omentum and ileal loops.

b) In pelvic congestion, pain is more frequent in the premenstrual stage; other times, after intercourse (especially anorgasmic) or caused by prolonged standing.

There may be a history of circulatory disorders and, on physical examination, evidence of varicose veins in the lower limbs or genital organs (vulva, vagina, uterus).

c) In benign neoplasms, in addition to pain, the woman may report the notion of the abdominal tumor, when it is large and becomes extrapelvic. The gynecological examination easily reveals ovarian or uterine tumors (fibroids) in most cases.

d) In malignant neoplasms, pain is a late symptom, so when it appears, its diagnosis is almost always easy, as it is accompanied by irregular bleeding, sometimes post-coitus in neck cancer.

e) In uterine prolapse - the exit of the uterus outside the vulva - discomfort during intercourse with the sensation of weight or pain in the hypogastrium, make up the clinical picture.

On physical examination, the diagnosis is almost always evident by simple inspection or straining to bear down.

f) The pain caused may be secondary to the use of a device: loop, ring, copper T", spiral. The diagnosis is established by the history of the appearance of pain after the placement of the device, which may be accompanied by bleeding and coitalgia.

The physical examination may be negative or pain may be found on palpation of the uterus during vaginal examination.

Semidiagnosis

Pelvic pain, both acute and chronic, can be caused by different gynecological causes:

1. Gynecological causes of acute pain:

Functional	Ovulation Primary dysmenorrhea
inflammation	Inflammation acute pelvic
Tumor	Complicated ovarian cyst Complicated fibroid
Hemorrhagic	ectopic pregnancy

2. Gynecological causes of chronic pain:

Functional	Pelvic congestion
inflammation	Chronic pelvic inflammation
Tumor	Uterine fibroid Ovarian tumor and cysts Cervical and uterine body cancer Pelvic endometriosis
Dystopian	Uterine prolapse
Due to foreign body	Intrauterine devices

Menstrual disorders

Concept

Menstrual disorders are understood as pathological deviations of menstruation outside of what is accepted as a normal menstrual cycle (5 days + 2 every 28 days + 7). The isolated variation of any month in the characteristics of menstruation, which is not repeated in the following months, has no semiological significance. For study and guidance, only cycle alterations that are repeated for three months or more should be considered.

A precise definition of the nomenclature of menstrual disorders must be used; In our country we use the following:

1. Quantity:

a) Hypermenorrhea (from hyper: more): increase in the daily amount of blood (more than 6 pads).

b) Hyponenorrhea (from hypo: less): decrease in the daily amount of blood.

2. Duration:

a) Polymenorrhea (from poly: a lot): increased duration of menstruation (more than 5 days).

b) Oligenomenorrhea (from oligo: little): decreased duration of menstruation (less than 3 days).

3. Periodicity:

a) Amenorrhea (a: privative particle): absence of menstruation for a period greater than 4-6 months.

b) Opsomenorrhea (from opso: elongated): rhythmically elongated cycles; menstruation every 5 weeks or more.

c) Proiomenorrhea (from proio: premature): the cycles are rhythmically short: menstruation every 3 weeks.

nas or less.

4. Combined:

a) Hyperpolymenorrhea (menorrhagia): increase in the amount and duration of menstruation.

b) Hypoligomenorrhea: decrease in the quantity and duration of menstruation.

c) Opsoligomenorrhea: lengthened cycles (in time) with a decrease in the duration (in days) of menstruation.

d) Metrorrhagia (metro: uterus; bleeding: flowing blood): irregular uterine bleeding independent of the menstrual cycle.

Semiogenesis or pathophysiology

Menstrual disorders can be caused by functional alterations or organic conditions:

uterine, ovarian, hypothalamic-pituitary or endocrine.

Let us remember that menstruation is the shedding (which occurs cyclically) of an endometrium that has undergone changes in proliferation and secretion, induced by estrogens and progesterone secreted by the ovaries. The maturation phases of the ovarian follicle originate when the follicle-stimulating hormone (FSH) first acts on the primordial follicle, which causes its maturation; Later, luteinizing hormone triggers follicular rupture or ovulation. In the follicular maturation stage, estrogens are produced, and at the end and after ovulation, progesterone appears, and estrogen production also persists. In the case of

If there is no fertilization, the ovary ceases its hormonal production and this drop is what causes endometrial shedding (menstruation).

When a process, functional or organic, alters any of these mechanisms, a menstrual disorder may occur.

The semiogenesis of amenorrhea will be assessed in a separate section.

In relation to the rest of the menstrual disorders, they are explained by alterations in ovulation (anovulation), in the production of estrogens (hypostrinism or hyperstrinism), of progesterone (aluteinism, hypoluteinism or hyperluteinism) or of androgens (virilism), sometimes they are associated with endocrine syndromes of another location (pituitary, thyroid, adrenal, pancreas) or with functioning tumors. of the ovary.

Semiotechnics

When faced with a menstrual disorder, a thorough and well-directed interrogation is the first thing.

One of the most useful investigations is diagnostic curettage, which should preferably be performed in the premenstrual phase. Sometimes, a curettage may be necessary in full bleeding, which serves as therapeutic and diagnostic purposes.

Among the common tests for diagnosing ovulation (see amenorrhea) we have:

a) Basal temperature curve.
b) Filancia and serial crystallization of cervical mucus.
c) Serial vaginal cytology.

semiography

When faced with a menstrual disorder, the time of appearance must be evaluated, if it is accompanied by other symptoms such as pain, fever, if it is liquid blood or with clots, if it is fresh (red) or retained (dark), etc. The gynecological examination also allows us to rule out organic causes of bleeding.

Of special significance is the condition called dysfunctional uterine hemorrhage, which frequently appears in the postmenarchal or pubertal stage and in the premenopausal stage. It recognizes the cause as anovulation with sustained hyperstrinism that prolongs the cycle; When menstruation appears, hyperpolymenorrhea occurs, which can lead to hemorrhage that motivates the patient's admission. In premenopausal women it can be associated with significant endometrial alterations (endometrial hyperplasia), which can be simple, cystic or adeno-

bushy

Semidiagnosis

1.Cycl Os	Ovulatory Anovulatory
2. Estrogen production	Normal Hypostrinism Hyperestrinism (includes feminizing ovarian Absent
3. Production I utel	Normal Luteal insufficiency Hyperluteinism
4. With androgenic production (rare)	Adrenogenital syndrome (adrenal hyperfunction) Achard-Thiers syndrome (diabetes of bearded women) Cushing syndrome (pituitary basophilic adenoma) Functioning ovarian tumors
5. With others al terations (rare)	Frölich syndrome (obesity of the lower half of the body, skeletal and sexual retardation, delayed puberty and oligomenorrhea) Laurence-Moon-Biedl syndrome (similar to Frölich, with multiple congenital malformations) Thyroid syndromes (hyperthyroidism, hypothyroidism)

Amenorrhea

Concept

The absence of menstruation is called amenorrhea. This is considered physiological before menarche or first menstruation, which in our environment occurs at the age of 12, on average. During pregnancy, amenorrhea is also considered physiological, as well as its maintenance, while breastfeeding, in many women.

Finally, the amenorrhea that is observed in women after past reproductive age (around 47 years) and is called menopause is considered physiological.

The difference should be highlighted between primary anenorrhea, which is one in which the woman has never had menstruation, as occurs in those of genetic cause, and the so-called secondary amenorrhea, in which the woman, after having had her menstruation for a certain time, presents amenorrhea; Those that have evolved four months or more (six months according to the WHO) should be considered as such for study.

Semiogenesis and semidiagnosis

Multiple causes can give rise to amenorrhea if the complex mechanism of menstruation and its endocrine interrelation are taken into account.

We will first mention the so-called anatomical factors that can be located at the level of the ovary, uterus, vagina or vulva; These factors can be congenital and acquired.

Congenital. Gonadal agenesis, uterine agenesis or hypoplasia, vaginal agenesis and imperforation of the hymen (cryptomenorrhea or occult menstruation).

Acquired. Castration, hysterectomy, destruction of the endometrium by exhaustive curettage, tuberculosis, radiation or severe inflammation (in the latter case the uterine walls may adhere firmly and the uterine cavity may totally or partially disappear due to synechiae) and. finally, post-traumatic cervical synechiae, or as a scarring consequence of acute endocervicitis, with retention of the menstruation product within the uterus (hematometra).

Alterations of the secondary gonads due to genetic disorders: gonadal dysgenesis (Turner syndrome), pure or mixed gonadal dysgenesis, true hermaphroditism, feminizing testicular syndrome and congenital adrenal hyperplasia.

The close interrelationship and balance of the endocrine system can be altered and lead to amenorrhea due to dysfunction (hypo- or hyperfunction).

Conditions of the pituitary gland, thyroid, adrenals, pancreas or ovaries can also cause amenorrhea.

The relationship of the pituitary gland with the diencephalon and the cerebral cortex explains the existence of psychogenic amenorrhea as a manifestation of nervous conditions: cases of psychosis, psychoneurosis, anorexia nervosa and particularly, in pseudocyesis or psychic pregnancy and in the so-called "inpatient" or hypothalamic (corticohypothalamic) amenorrhea.

Finally, the so-called constitutional factors are grouped within the causes of amenorrhea, some of nutritional origin: poor nutrition due to defect (anemia) or poor nutrition due to excess (obesity); other factors of infectious origin: tuberculosis, leprosy and, finally, the action of some medications (especially psychotropic drugs and hormones).

Semiotechnics

It is extremely important to carry out a detailed anamnesis taking into account the multiplicity of causes and their relationship with different devices or systems, which is why we must insist on: age, nationality, social status, family pathological history (tuberculosis, leprosy, consanguinity, genetic diseases), personal pathological history (endocrine-metabolic, nutritional, nervous conditions, medications used), gynecological history (menarche, menstrual formula, sexual relations). sexual intercourse, pregnancies, gynecological interventions or examinations), accompanying symptoms, evolution of the clinical picture and previous treatments.

In the general physical examination, the following will be assessed: alterations in height, distribution of adipose tissue, muscle development, characteristics of body hair, size of the thyroid gland and alterations in the abdomen; in the examination of the genital system: characteristics of the external genitalia (inspection) and genital hair; in the speculum examination: vaginal and cervical color, moisture and trophism, vaginal contents, cervical mucus; on vaginal examination: size and elasticity of the vagina, characteristics of the uterus (size, shape, position, mobility), and characteristics of the ovaries (especially size and position). If necessary, hysterometry will be performed.

During the breast examination, breast development and changes in sensitivity, anatomical alterations (due to palpation) and functional alterations (galactorrhea) will be assessed.

Many times a positive diagnosis can be established by history and physical examination; In others, research is necessary.

semiography

In the study of primary amenorrhea, it is possible to find congenital causes through history and physical examination.

We must insist on the evaluation of secondary amenorrhea from 4-6 months onwards, since this prudent period ensures the differential diagnosis with pregnancy - an entity that causes more than 90% of amenorrhea - and also with some opsomenorrhea (ovarian amenorrhea), which frequently evolve to spontaneous resolution, as is the case of the existence of a persistent follicle, a cyst. functioning follicular or luteal cysts. In some cases, these causes may persist for longer.

Likewise, a special history of the genital tract is very important in the diagnosis of secondary amenorrhea: the way in which the amnenorrhea occurred, whether it was progressive or abrupt, and, ultimately, its relationship with childbirth, postpartum hemorrhage, abortions, genital infections, uterine curettage or surgical interventions.

Leucorrhoea

Concept

Also called "vaginal discharge" or simply "discharge", it is the release to the outside of the increased and modified vaginal contents, the result of secretions or transudations from the genital organs. Other authors insist on specifying that any material that flows from the genitals and is not menstruation or blood should be considered leucorrhoea. This symptom constitutes the first cause or reason for consultation in Gynecology.

Semiogenesis

The genital tract is kept moistened with the product of secretions from the glands in its different segments (tubal, uterine body, neck, vagina, vulva), but it undergoes physiological variations throughout the menstrual cycle.

Usually the increase in one of these secretions or the transudation produced by an inflammatory process in some of these segments, especially in the cervix and vagina, causes this material to flow to the outside, giving the clinical manifestation of leucorrhoea. It may also be due to the mechanical action on the endometrium of an intrauterine device or to the zonal necrosis of an advanced infiltrating cancer of the uterine cervix. Delayed postmenstrual healing of the endometrium due to hypostrinism can produce short-lived postmenstrual leucorrhoea.

Semiotechnics

The woman may report "stains" on her underwear, more or less abundant, which she can show in the office, and when performing the physical examination we can find a material that is usually liquid, lumpy and even purulent, which wets part of the vulva and the vaginal introitus; When questioned, she reports having washed or cleaned herself before going to the consultation. On other occasions, she goes at a time when she does not have flow, since it undergoes cyclical variations. Therefore, it is very important to call the patient back for examination at the time when the symptom is present and without cleaning or previous washing, to be able to observe all the details of quantity, color, smell, physical appearance, etc.

If the discharge is observable, we can take a sample to perform a direct study in fresh (vaginal exudate) or, after extension and desiccation, a stain using the Gram technique or also, to perform culture in special media according to the germs that cause it.

Semiography and semidiagnosis

There may be discharge without other accompanying symptoms and without organic lesions of the genital system; In this case it is due to the hyperproduction of cervical mucus, which appears abundant, very thin, clean, in the final days of the estrogenic phase and in the ovulatory phase, that is, between the tenth and fifteenth days of the cycle.

This flow can be abundant and so evident that it even runs towards the thighs in the standing position.

Increased mucus can also be caused by a more or less extensive ectopia of endocervical epithelium in the exocervix.

The body discharge can occur in the immediate days after menstruation, in the form of bloody spots, which some authors consider normal if they are scarce and only last 2 or 3 days, but abnormal if they last longer and, above all, if they occur in greater quantity; In these cases it may be the expression of an estrogen deficiency in the initial phase of the cycle that delays the healing of the endometrium or also the result of chronic endometritis (rare).

The discharge of infection by Candida albicans (moniliasis) and other yeasts is usually white, lumpy, adherent to the skin of the lips of the vulva and vaginal wall, accompanied by itching that can become very intense and add scratching lesions on the vulva (fig. 70.14). When these lumps break off, they can leave a reddish, superficially ulcerated surface. This flow is more frequent in pregnant women, diabetics and obese women; It may also begin after a course of treatment with broad-spectrum antibiotics.

The discharge produced by Trichomonas vaginalis almost competes in frequency with moniliasis. It can be yellow or greenish, very fluid, foamy; especially in acute cases; at other times, watery, yellowish, especially in chronic or relapsing cases (fig. 70.15). Sometimes it can be mucopurulent.

The discharge of trichomoniasis can produce vulvar itching, although less generalized than in moniliasis, and a burning or burning sensation referred to the most posterior area of the vulva. There may be dyspareunia or stench reported by the patient and discomfort caused by the examination (vaginal examination and speculum examination).

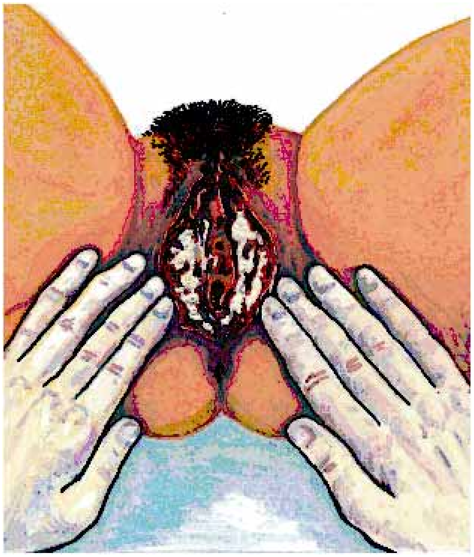
In very acute cases, cervicovaginal petechial stippling called "strawberry vagina" may be seen.

The discharge of gonorrhea is frankly purulent, fetid; It may be accompanied by vaginal itching, burning and dysuria. Occasionally, accompanying acute bartholinitis may occur or be the main manifestation. On physical examination one can find: purulent endocervicitis, signs of urethritis, eschenitis, acute or chronic salpingitis with large tubal dilations and even pelvic peritonitis.

Nonspecific leucorrhoea, as the name His indicates, does not present the typical characteristics of the three described above nor its characteristic accompanying symptoms (Fig. 70.16).
It is whitish or slightly yellowish, less abundant and frequently evolves in a chronic, relapsing or cyclical form. Upon examination of the fresh exudate and with Gram stain, no monilias, trichomonas or diplococci were observed; Other germs can be found such as:

staphylococcus, streptococcus, Escherichia coli. diphtheroid bacilli, enterococci and Hemophylus vaginalis; Sometimes it is necessary to perform a culture of the secretion to determine the causative germ and an antibiogram to guide antibiotic treatment. This leucorrhoea is very often associated with chronic cervicitis and presents frequent recurrences. In the speculum examination, typical lesions of periorificial redness due to ectopia may be observed.

Fig. 70.14 Leucorrhoea in moniliasis.



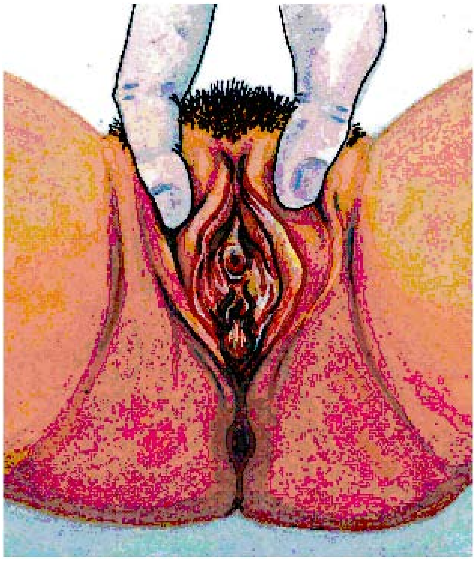
of the endocervical epithelium, Nabothian eggs, whitish areas of re-epithelialization and cervical tears.

Vaginal discharge can be an accompanying symptom of acute pelvic inflammation (metroadnexitis), with high fever, intense pelvic pain and general manifestations: general malaise, asthenia, anorexia and even dizziness and vomiting. It can also be an accompanying symptom of chronic pelvic inflammation, where the predominant symptom is pelvic pain.

We relatively frequently see the discharge produced by the use of contraceptive intrauterine devices, which is generally postmenstrual, fluid, pink or slightly bloody. When it lasts a few days it is a lesser evil that many women prefer to endure before running the risk of an unwanted pregnancy; If it is very abundant or continuous, it requires a more in-depth analysis to decide the behavior that should be followed.

As mentioned in semiogenesis, a cancer of the cervix or endometrium can sometimes produce leucorrhoea, which is almost always bloody (like "meat washing water"), fetid (due to the organic remains produced by tissue necrosis) and accompanied by bleeding, caused by intercourse. The diagnosis will be made evident by the observation of the malignant lesion in the speculum examination or by a diagnostic curettage.

Fig. 70.15 Leucorrhoea in trichomoniasis.



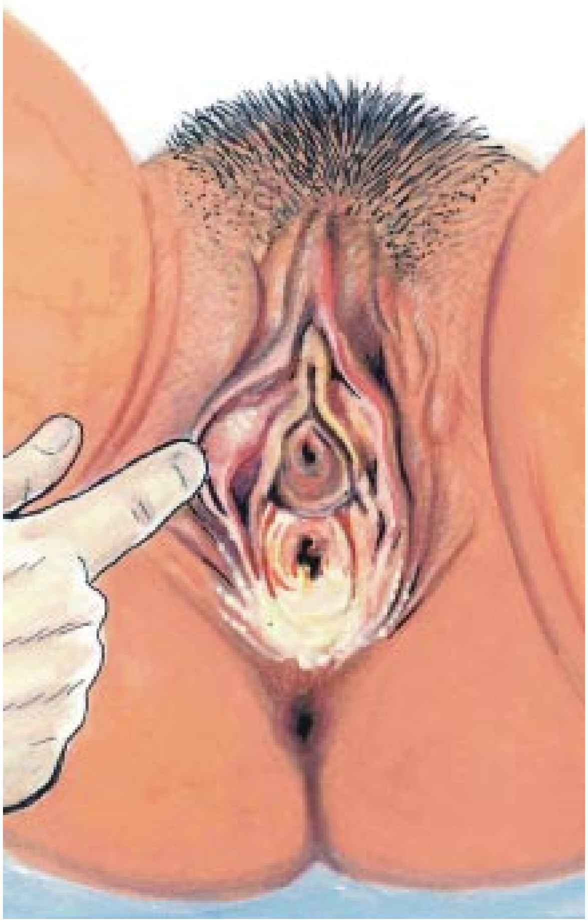


Fig. 70.16 Nonspecific leucorrhoea.

ALTERATIONS IN THE PHYSICAL EXAMINATION

After a good interrogation, a thorough physical examination is required, which includes, of course, the gynecological examination, but which must begin with the general and regional physical examination.

In the regional examination, examination of the breasts is important to detect any alteration, while inspection and palpation of the abdomen guide and allow the tumor processes to be specified.

Breast semiology

Anatomy, physiology, and breast examination and self-examination were covered in volume 1 (see Section I, Chapter 4). Now we will briefly offer the most frequent conditions and signs to take into account in the physical examination, which will be studied in greater detail in the texts of other subjects, such as Surgery.

Acute mastitis is associated with lactation, but can occur at any time and at any age. Irritation and abrasion of the nipple and breast skin are noted It is reddish. It is usually accompanied by systemic signs, such as fever and tachycardia.

Signs of retraction include depressions, wrinkles or folds in the skin and changes in the contour of the breast or nipple. They can only become evident with changes in position or with palpation of the breast. They may be secondary to scar tissue formation (fibrosis) or be a warning sign of possible breast neoplasia.

Nipple inversion is considered normal if it has been present for a long time. If it is newly developed, it is associated with fibrosis and malignancy.

Prominence of the venous circulation is also associated with cancer, if it is unilateral and the increase in the venous pattern is localized.

In the orange peel sign the skin is hard, immobile and has the appearance of an orange peel, with enlarged pores. Skin discoloration may occur. It can be seen in the areola. It is associated with cancer and is caused by interference with lymphatic drainage of the

mother.

In Paget's disease (mammary duct malignancy) the early signs are: erythema of the nipple and areola, and the late signs: thickening, cracks and erosions of the nipple and areola.

palpable masses

1. Fibrocystic disease (benign fibrous cysts). It appears as a solitary mass or as multiple masses. They are usually sensitive (painful), round in shape, elastic or firm in consistency, and mobile. Referral to a specialist and biopsy should be indicated for definitive diagnosis.

2. Fibroadenoma (benign breast neoplasm). It usually appears as a solitary mass. It is usually not painful. It can be rounded or lobular. It is firm, mobile, not fixed to the breast tissue. Referral and biopsy are indicated for definitive diagnosis.

3. Breast cancer mass. It usually presents as a single mass, in one breast. It is usually not painful either. It has an irregular shape and a firm, hard consistency, immersed in the surrounding tissue. Referral and biopsy are also indicated for definitive diagnosis.

Increase in volume of the uterus

Concept

The normal morphological data of the uterus were described in Section I of volume 1, in Chapter 20. If we start from the knowledge of the average size of the uterus

(8 cm x 4.5 cm x 3 cm in longitudinal, transverse and anteroposterior diameter, respectively), we must consider its increase in size or volume if the figure found is above these data. In nulliparous women, especially those under 18-20 years of age, the uterus may be somewhat smaller than the average size; It increases later, due to pregnancies, especially if they reach the last trimester. In multiparous women, the size of the uterus may be slightly larger than what is considered average and then we speak of a "large multiparous uterus."

Semiogenesis or pathophysiology

Pregnancy. The existence of an egg that implants in the secretory endometrium and the hormonal production (chorionic gonadotropin) of the ovular chorion, with the consequent maintenance of the production, by the corpus luteum, of progesterone (and estrogen, in a lesser quantity), produce hyperplasia and hypertrophy of the myometrium. The growth of the egg progressively distends the walls of the organ, which is softened by the action of progesterone.

Later, in the second trimester, the placenta maintains hormonal production until the end of pregnancy.

tion.

Benign malignant neoplastic processes. They enlarge and deform the uterus due to the growth of the tumor.

Severe inflammatory processes. They give rise to acute pelvic inflammation: metritis and metroadnexitis. Active inflammatory congestion and accumulation of purulent secretions produce a discrete or moderate increase in uterine size. If the cervical canal is obstructed, pus accumulates inside the uterine cavity or pyometrium.

Inflammatory or traumatic cervical stenosis or synechiae. It can cause the accumulation of the product of endometrial shedding within the uterine cavity, for months or years (hematometra), with a progressive increase in uterine size.

Uterine endometriosis. The ectopic location of endometrial tissue in the thickness of the uterine muscles, which may or may not have communication with the cavity, also produces an increase in the size of the uterus.

Semiotechnics

The anamnesis allows orienting the diagnosis, taking into account the general data and, in particular, those referring to the genital tract: menarche, menstrual formula, previous pregnancies (births, abortions, ectopic pregnancies, cesarean sections), gynecological operations, as well as present symptoms.

By inspection of the abdomen, the increase in volume can be seen, especially in eutrophic and thin patients, which causes a prominence of the hypogastric area that reaches the navel and surpasses it in cases of pregnancy.

A measurement of the uterine height can be taken, from the upper border of the pubis to the uterine fundus, which has value in the diagnosis of gestation time and in the assessment of intrauterine growth of the fetus (fig. 70.17). Obesity can modify these measurements.

By palpation we can determine the fetal parts during pregnancy: head, back, buttocks, limbs, and by auscultation the fetal focus is heard with a greater frequency than that of the mother, whose pulse is taken simultaneously.

If the uterus does not exceed 10 or 12 cm in diameter, its enlargement cannot be evident by inspection or abdominal palpation.

The speculum examination provides important elements for the diagnosis in pregnancy: the gravidarum bluish discoloration of the exocervix and vagina; the compact cervical mucus, very thin and crystallizable, in addition to the intensity with which it is stained with the iodinated coloration of the Schiller test.

Only in a few other cases, such as neck cancer and cervical fibromas, will the speculum examination provide data of interest.

Vaginoabdominal examination (bimanual) is the examination that allows us to better specify the increase in size of the uterus, even when it is very discreet, as well as its changes in shape, mobility, location of nodules, consistency, etc.

In the virgin woman, a rectal or rectoabdominal examination will be performed.

The touch combined with abdominal palpation serves to specify the increase in volume of the uterus due to other non-physiological pathologies, such as benign or malignant neoplasms.

Other methods like:

Hysterometry, in the pyometrium and in the hematometra.

Laparoscopy, in tumor or inflammatory processes.

Radiology, in the third trimester of pregnancy.

Ultrasound, in pregnancy at any stage and in supposedly tumorous processes, can be of great help.

semiography

The extraordinary value of the interrogation should be highlighted in the careful and detailed collection of all the symptoms and signs, as well as the chronological order of all the facts that may be related to the diagnosis and which frequently allows establishing a presumptive diagnosis with a high probability of being positive.

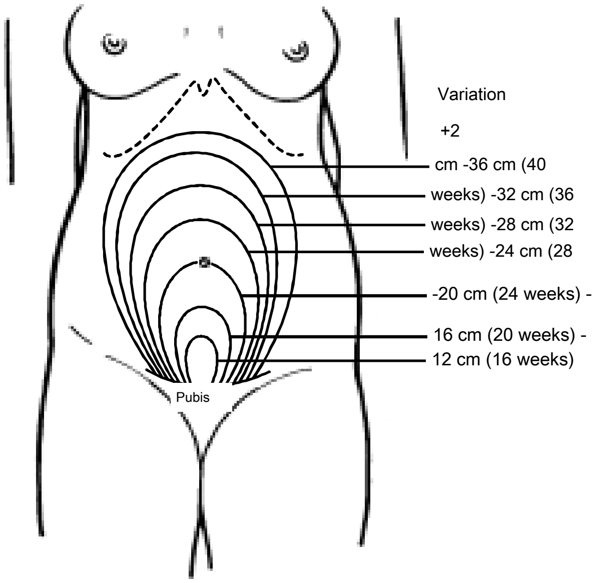


Fig. 70.17 Measurement of uterine height in centimeters and its approximate correlation with gestation time in weeks, counted from the first day of the last menstruation, according to the International Federation of Gynecology and Obstetrics (FIGO).

In pregnancy, the most important sign is amenorrhea (which can be hidden by the woman), which has much more value if the patient is eumenorrheic. It may be accompanied by digestive symptoms, sialorrhea, nausea, vomiting, anorexia, rejection of certain odors and flavors, character changes, drowsiness, breast tension, chloasma and pigmentation of the abdominal midline, which allow establishing the presumptive diagnosis, which is reinforced by breast signs: increased pigmentation of the areolas and nipples, development of Montgomery's tubercles and presence of colostrum in the moms.

The probable diagnosis of pregnancy is based on the increase in uterine volume, changes in its shape (globulous) and consistency (softened, especially in the isthmus), purplish discoloration of the neck, compact cervical mucus, and detection of an intense color in the Schille test.

Late diagnosis includes palpation of the fetal parts, appreciation of fetal movements, auscultation of the fetal focus and radiographic demonstration, or better by ultrasound, of the fetal skeleton.

In endometriosis (location of endometrial tissue outside the uterine cavity) hyperpolymenorrhea, secondary dysmenorrhea (which increases progressively), sterility, dyspareunia (coitalgia) and the uterus is enlarged, irregular and hard may occur.

Fibroid, which is the most common benign tumor in women, can occur asymptomatically or produce symptoms due to compression of neighboring organs or bleeding (hyperpolymenorrhea or bleeding).

intermenstrual) in relation to their anatomical location: intramural, subserous or submucosal (fig. 70.18).

Its complications (torsion and necrobiosis) can give rise to acute symptoms of intense pain and general condition tone.

Malignant neoplasms, usually, when they are evident on physical examination, carry symptoms for a long time: postmenopausal bleeding (endometrial adenocarcinoma), bloody leucorrhoea, bleeding from intercourse (neck cancer), weight loss and pain.

In acute pelvic inflammation there is frequently a recent history of childbirth, abortion, gynecological examination or intervention, from which the clinical picture can occur suddenly or progressively.

The condition begins with more or less intense pain, which limits the woman's activities, high fever (38-40 °C) with chills, tachycardia and leukorrhea (sometimes fetid), general deterioration and sometimes dysuria, nausea or vomiting. The physical examination, increased erythrocyte sedimentation rate and blood count with leukocytosis and left deviation allow a positive diagnosis to be established. In the case of pyometrium, the uterus will be enlarged, soft and retentive and when passing a hysterometer, the pus flows out, confirming the diagnosis.

In the hematometra, the increase in volume of the uterus, together with amenorrhea from a known event: abortion, curettage, acute metroadnexitis or cervicitis, electrocoagulation or conization, vaginal operation, etc., establish the presumptive diagnosis, which is confirmed with the permeabilization of the neck and the exit of the dark and thick contents of the uterine cavity.

Semidiagnosis

The most important causes of increased uterine volume are:

and pregnancy	Most frequent cause
Tumors	Benign (fibroids, fibroids, fibromyomas) Malignant (squamous cell carcinoma of the neck, endometrial adenocarcinoma)
Inflammation	Acute pelvic inflammation Pyometrium
Dystopia	Uterine endometriosis
hematometra	Obstructive

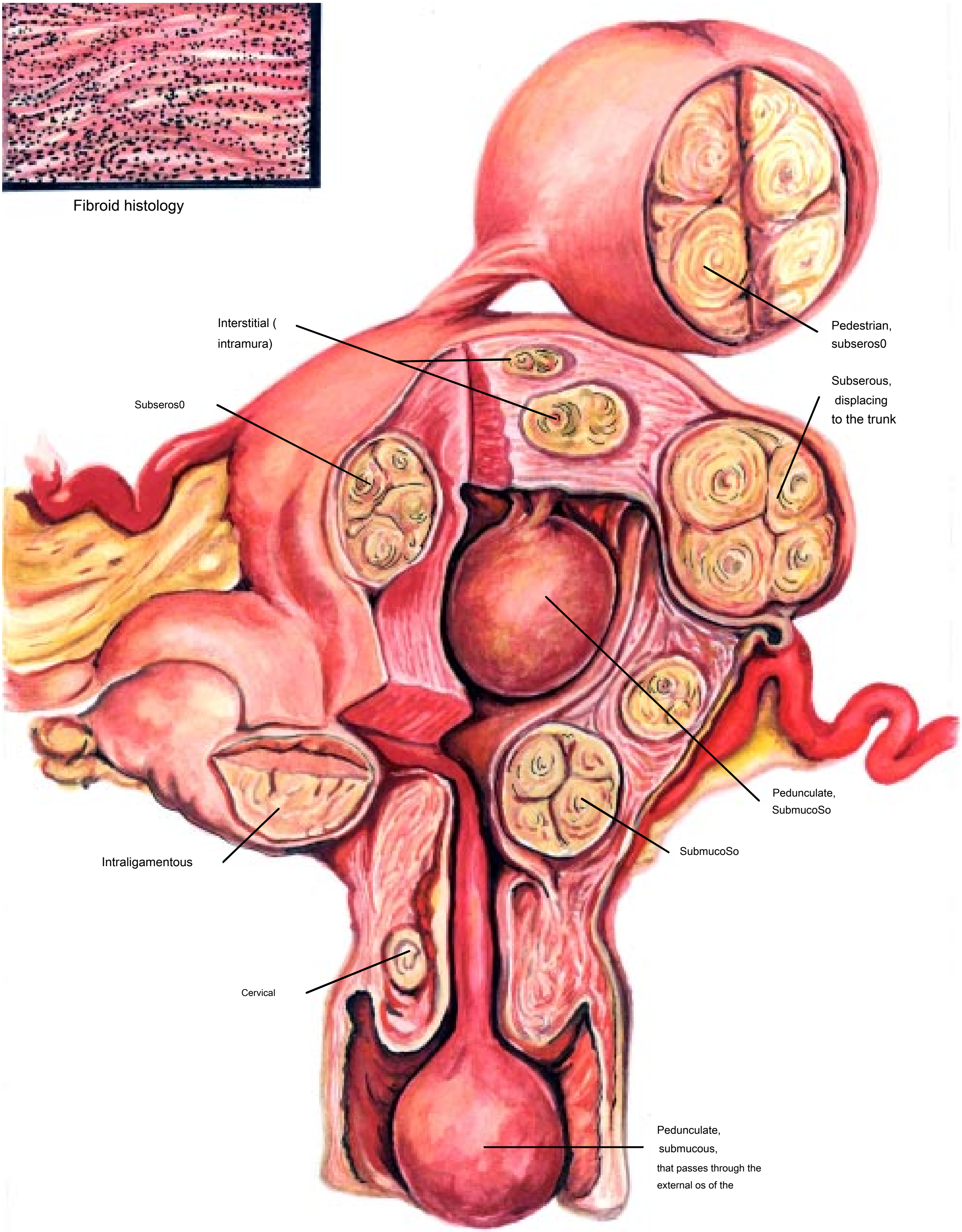

Fibroid histology
Interstitial (
intramura)
Pedestrian,
subseros0
Subserous,
displacing
to the trunk
Subseros0
Pedunculate,
SubmucoSo
SubmucoSo
Intraligamentous
Cervical
Pedunculate,
submucous,
that passes through the
external os of the

Fig. 70.18 Uterine fibroids.

Increased volume of the tubes

Concept

The tubes are organs with a very simple structure, thin walls and a very narrow light conduit (1-4 mm); Therefore, we will say that there is an increase in volume of the tubes, when on palpation they are above the indicated measurements. This is generally due to the increase in content in its cavity.

Semiogenesis and semidiagnosis

Infections by pyogenic bacteria can acutely affect the tubes (salpingitis) and sometimes, when the uterine orifice and abdominal ostium are occluded, pus accumulates in them (piosalpinx or pyosalpinx) with progressive dilation, causing elongated, pudgy tumors that can easily reach a diameter of 3 or 4 cm and less. frequently, 8-10 cm (fig. 70. 19).

Sometimes, the process also involves the ovary and gives rise to a tub0ovarian abscess or is part of an extensive process of acute pelvic inflammation (pelviperitonitis).

In order of frequency, it is followed by the tubal location of an ectopic pregnancy, which will grow progressively until the tube fissures or ruptures, producing an acute abdomen due to internal bleeding and, less frequently, if it is located in the ampulla, near the fimbria, it may be expelled into the abdomen and give rise to tubal abortion (fig. 70.20).

Neoplastic processes in the tubes are extraordinarily rare, truly exceptional; We can mention fibroma (benign) and adenocarcinoma (malignant).

Tubal localization of ectopic endometrial tissue, known as endometriosis, can cause an increase in the volume of the tubes, which grow progressively and reach large dimensions due to the formation of a hematosalpinx.

Semiotechnics

The anamnesis, the general examination, the gynecological examination (especially the bimanual examination), the ultrasound and the laparoscopy are the elements through which we will arrive at the diagnosis, most frequently.

Puncture of the Douglas cul-de-sac can help in the diagnosis of complicated ectopic pregnancy (ruptured or fissured).

Semiography

In relation to pregnancy, acute pelvic inflammation and endometriosis, see the semiography of the increase in uterine volume, in this same chapter.

In salpingitis we will find a slight increase in volume of the tubes, which are painful when moving the uterus and palpating them. In the case of a pyosalpinx or a tube-ovarian abscess, we will find intensely painful tumor masses, of a rather soft consistency and which are accompanied by a great deterioration in the general condition and peritoneal reaction.

The inflammatory pathology presents with fever, tachycardia, accelerated erythrocyte sedimentation rate, leukocytosis with left deviation, and by performing the abdominal or Douglas puncture we can extract pus. We can also see it when performing laparoscopy, which also shows the image of the entity that causes it.

Tumors can be observed by ultrasound, as long as they have diameters greater than 4-5 cm.

The presence of pregnancy in the tubes (tubal ectopic pregnancy) is more common in the ampullary area, less common in the isthmus.

This pregnancy can cause rupture of the tube (as frequently occurs when it is isthmic) or tubal abortion can occur, especially when the implantation is ampullary. The symptoms can be varied, which is why this pregnancy is sometimes called "the great simulator", although it is classically accompanied by amenorrhea or hypoligomenorrhea and pain in the hypogastrium (sometimes dull and intense).

When there is blood in the abdominal cavity (whether due to abortion or rupture), peritoneal and phrenic irritation appears (giving pain at the shoulder level); In addition, there may be signs of shock, depending on the amount of bleeding.

The presence of endometrium (endometriosis of the tube) responds to theories of uterine endometriosis.

This disease is characterized by a classic triad: menstrual pain (dysmenorrhea), pain during defecation, pain during intercourse (dyspareunia), which increases in intensity gradually and progressively. To touch, parauterine tumors can be seen, which may or may not be accompanied by bluish nodules in the vagina. Laparoscopy and ultrasound help define the entity, although sometimes the diagnosis is made by performing a laparotomy.

Increase in ovary volume

Concept

The increase in ovary volume should be considered based on its usual measurements: 3-5 cm in greatest diameter, 2-3 cm in transverse diameter and 1-2 cm in thickness. When the increase is discrete and does not exceed 6 cm, it should be evaluated conservatively and the examination repeated with an interval of one month, if there are no acute symptoms or the increase is only an occasional finding in the physical examination, especially when a resistant consistency is appreciated to the touch.

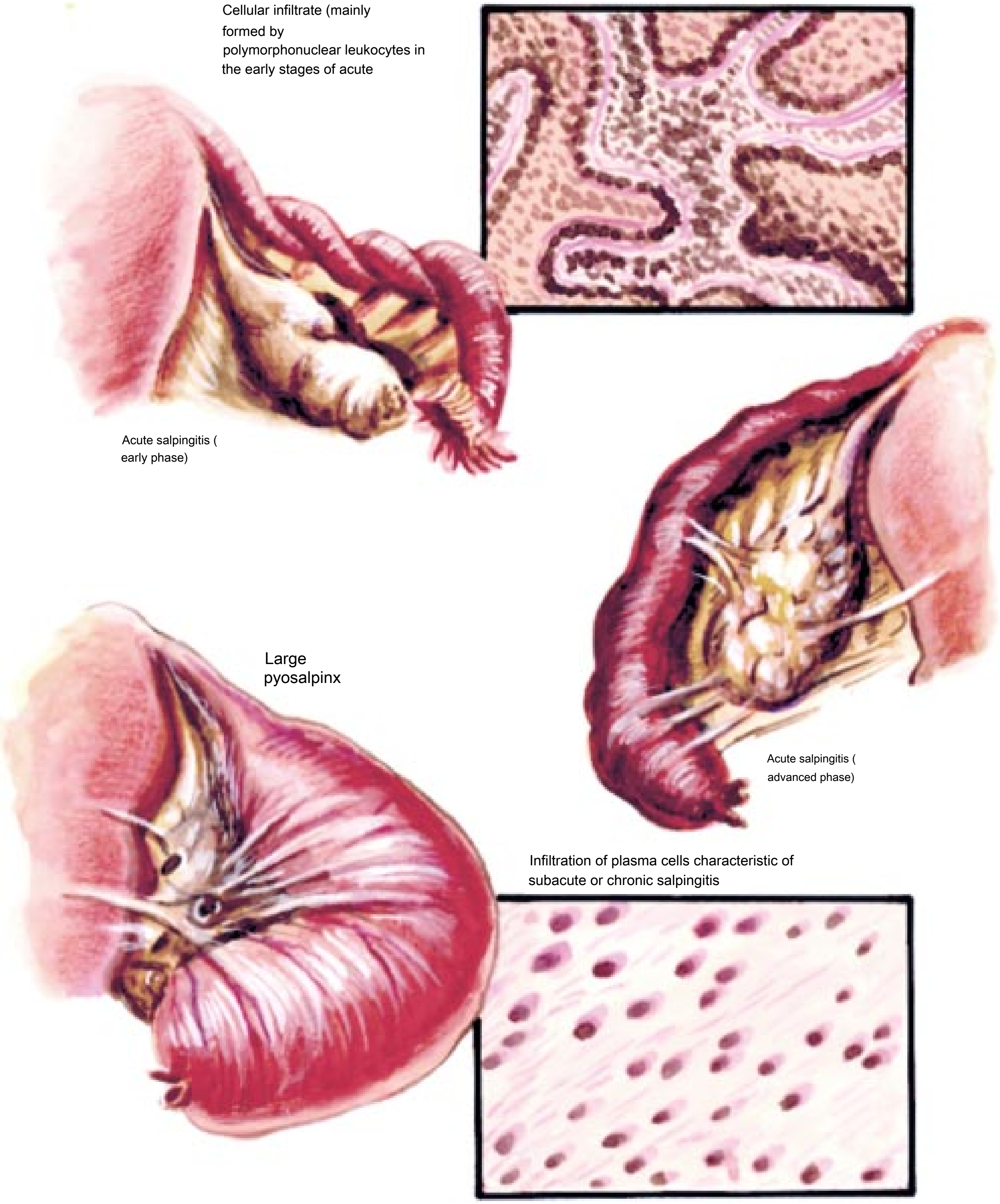

Cellular infiltrate (mainly formed by polymorphonuclear leukocytes in the early stages of acute
Acute salpingitis (early phase)
Acute salpingitis (advanced phase)
Large pyosalpinx
Infiltration of plasma cells characteristic of subacute or chronic salpingitis

Fig. 70.19 Acute salpingitis. Piosalpingex.

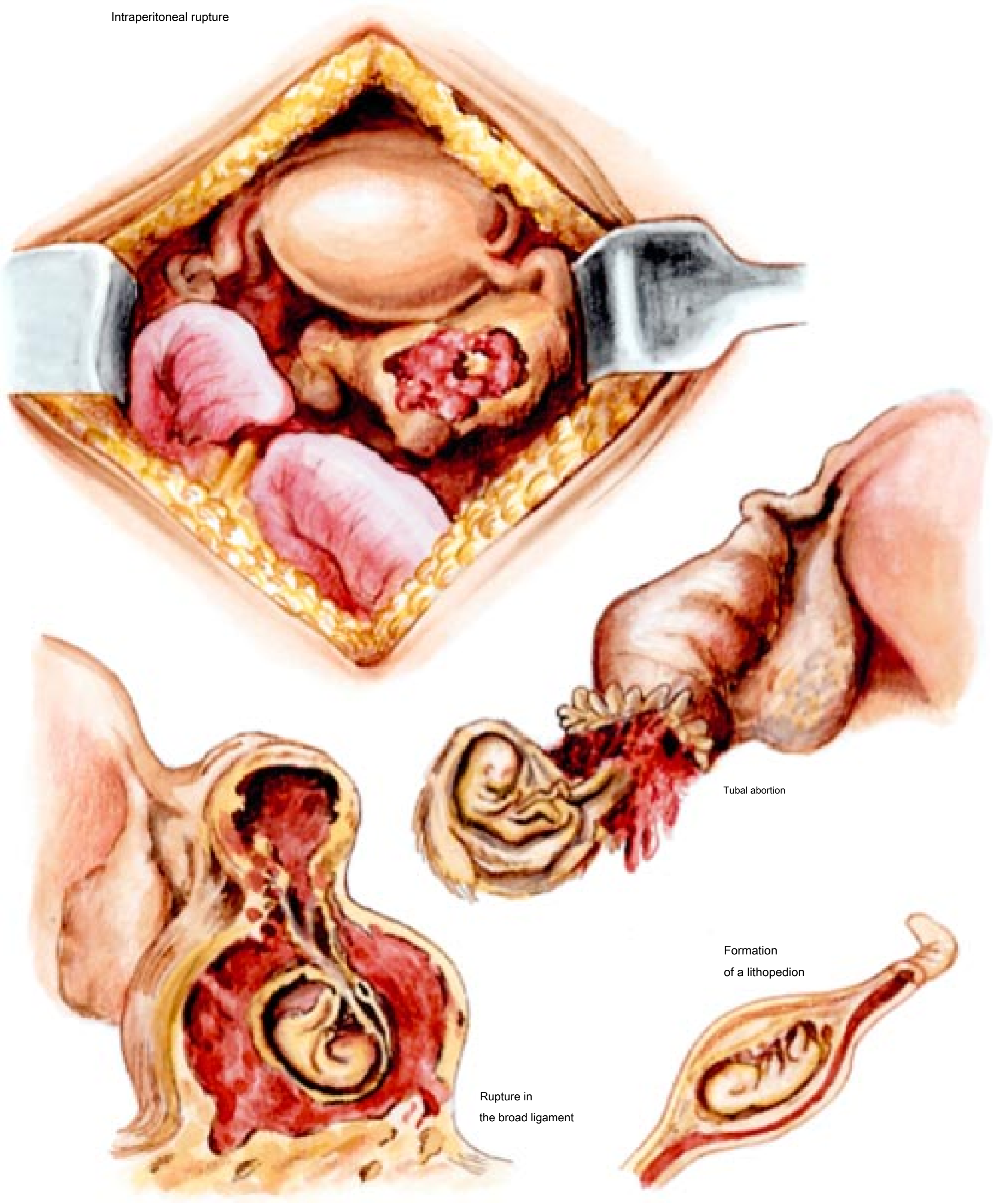

Intraperitoneal rupture
Tubal abortion
Formation
of a lithopedion
Rupture in
the broad ligament

Fig. 70.20 Ectopic pregnancy.

Semiogenesis and semidiagnosis

The increase in the normal structures of the ovary (follicles, yellow body) above usual can give rise to tumors (increase in volume and changes in consistency), more frequently cystic, which are known as "non-neoplastic ovarian tumors and which can be considered functional retention cysts.

The ovary of so-called Stein-Leventhal syndrome or polycystic ovarian disease and ovarian hyperthecosis (non-neoplastic) can be included in this group.

Other authors also consider the tumors produced by tube-ovarian inflammation and endometriosis in the previous group, which we explain separately to maintain the same schematic structure, followed by the increase in volume of the uterus and tubes.

Exceptionally, an abdominal pregnancy involving ovarian implantation can be seen.

Ovarian neoplasms present great variability. This is the organ of the economy where the greatest variety of them can originate, but for their better understanding by the general practitioner we will use the clinical classification of ovarian tumors, which divides them into:

1. Small and large.
2. Cystic and solid.
3. Benign and malignant.
4. Functioning and non-functioning.

Most frequent neoplasms

Benign cystic	Serous cystadenoma and mucinous Dermoid cyst or benign teratoma
benign solids	Fibroma and Brenner tumor
Evil cystic	Cystadenocarcinoma mucinous and seroSo Malignant teratoma
Just gone bad	Adenocarcinoma, endomeroid carcinoma and mesonephroma Less common: chorioepite-lioma, sarcoma, lymphoma, melanoma
Functioning (from low bad ignity)	Dysgerminoma and luteinized granulosa (feminizing) Arrenoblastoma, of adrenal remains and hilar adenoma (virilizing)
Metastatic	Krukenberg tumor

Semiotechnics

We must say that the anamnesis, the general examination, the gynecological examination (especially the bimanual examination), the ultrasound and the laparoscopy are the elements that should be used, and through which the positive diagnosis is reached most frequently. Hormonal tests are used in functioning or hormone-producing tumors.

Semiography

Non-neoplastic ovarian tumors are frequently oligosymptomatic or asymptomatic and constitute a finding during the gynecological physical examination. The persistent follicle, which maintains intense estrogenic activity, can produce opsomenorrhea with functional evidence of estrogenic action: elevated estrogens in blood and urine, functional cytology with a high index of superficial karyopyknotic cells, cervical mucus with filance of up to 8-10 cm, and intense iodine uptake in the Schiller test.

In the hematoma of the corpus luteum and in the so-called luteal cyst, the symptoms can suggest an ectopic pregnancy and the adnexal tumor can be misleading, and the differential diagnosis must be established by pregnancy tests and laparoscopy, to avoid unnecessary operations.

In Stein-Leventhal syndrome, sterility, oligoamenorrhea and menorrhagia are common, and hirsutism and obesity are not uncommon (Fig. 70.21).

Adnexal and tube-ovarian inflammatory processes, especially the abscess of this location, are usually accompanied by an intense general syndrome: high fever (38-40 °C), chills, tachycardia and general deterioration, which accompany intense, sustained pain in one of the iliac fossae. Hematic alterations (leukocytosis with left shift) and increased erythrocyte sedimentation rate help guide the diagnosis.

In endometriosis, the pain is initially a dysmenorrhea, with a progressive increase that can later become continuous, when the tumor masses increase in size; It can be accompanied by sterility and dyspareunia.

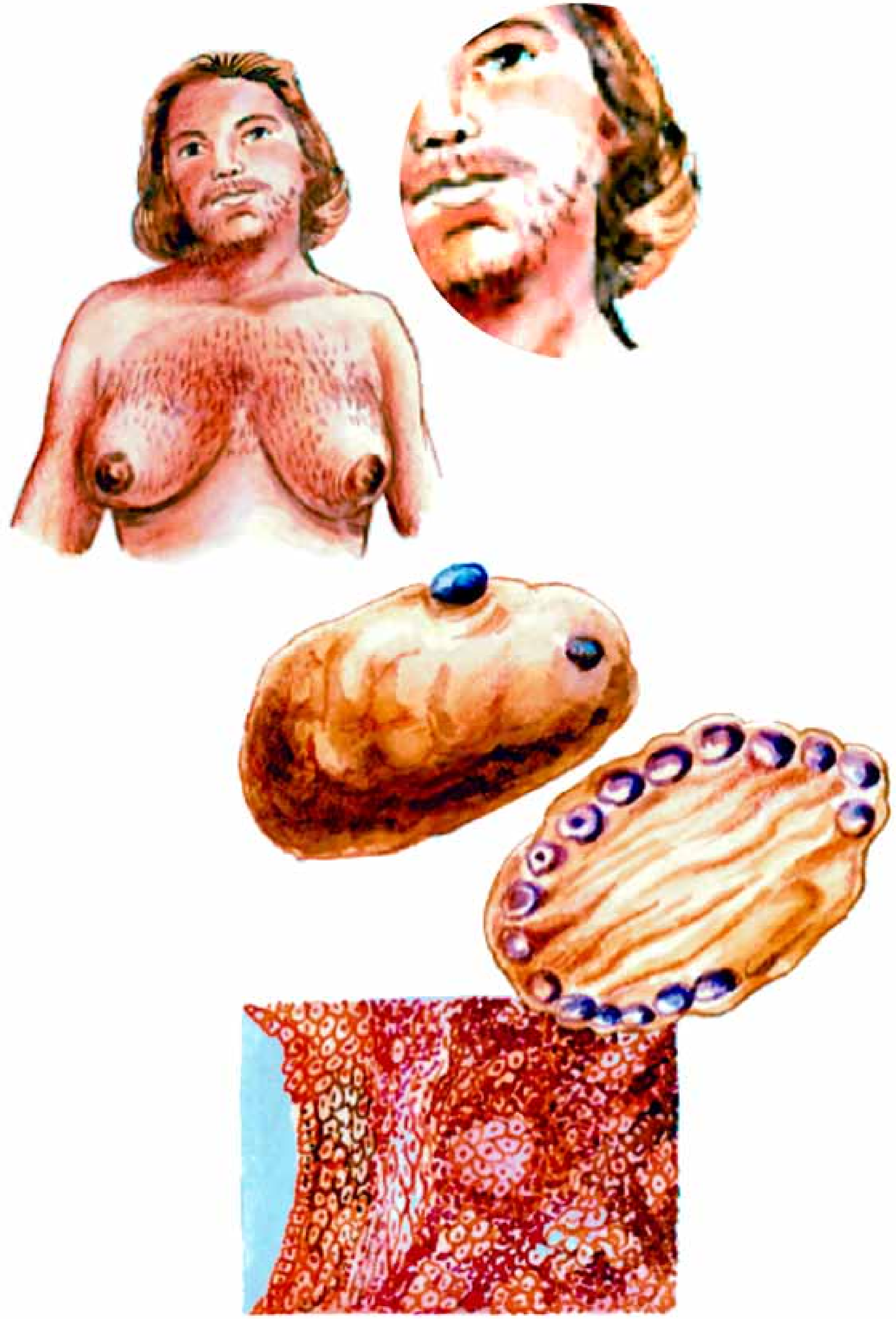


Fig. 70.21 Stein-Leventhal syndrome.

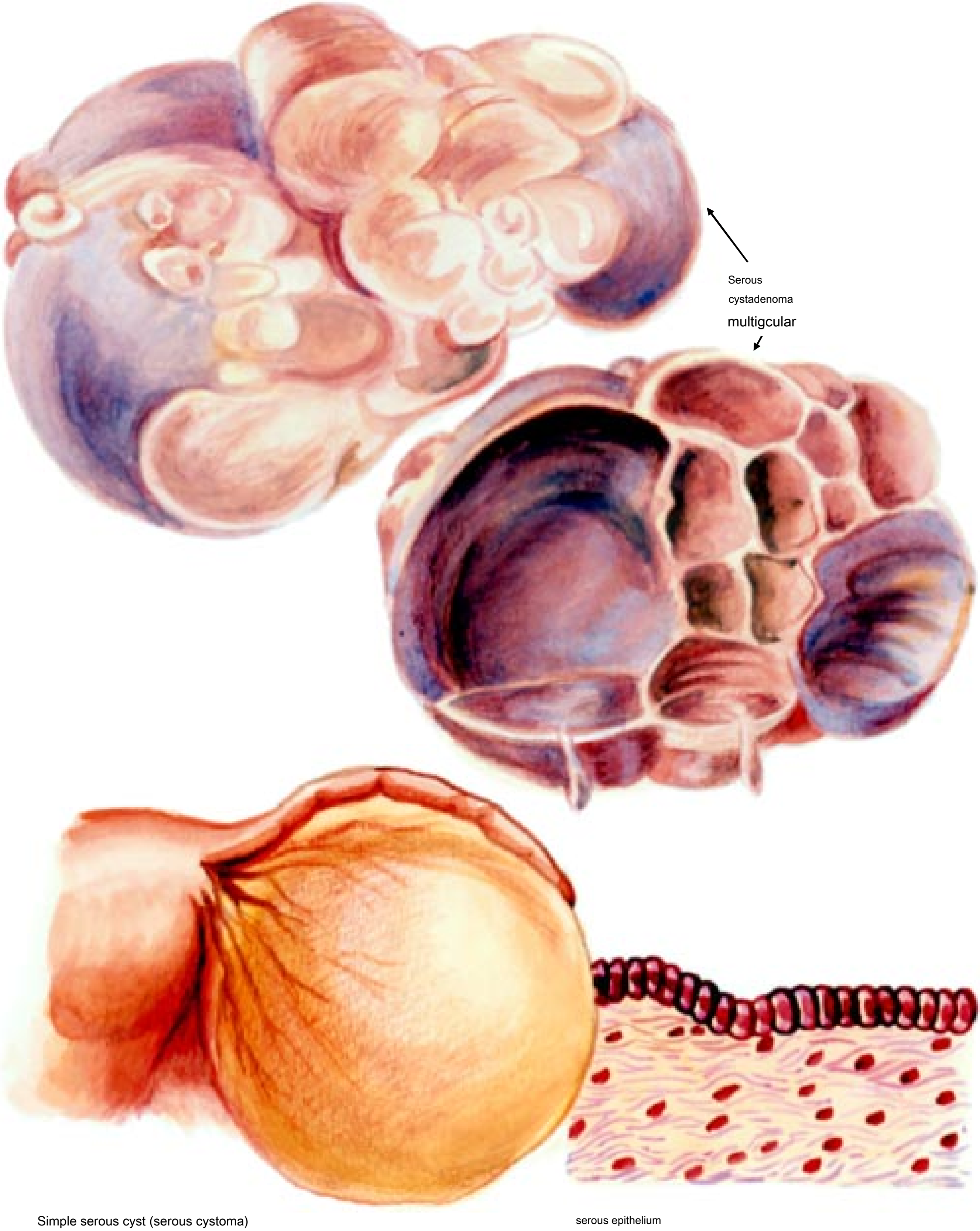

Serous
cystadenoma
multigcular
Simple serous cyst (serous cystoma)
serous epithelium

Fig. 70.22 Ovarian cyst.

In ovarian pregnancy, the progressive increase in volume of the tumor, amenorrhea and positive pregnancy tests, together with a normal or slightly enlarged uterus, justify the indication of a laparoscopy, which must be conclusive in the diagnosis.

Small ovarian cysts (up to 6 cm) should be monitored during their evolution; In practice we have seen cysts of 8 and 10 cm disappear spontaneously, confirming their non-neoplastic nature (fig. 70.22). If they evolve by growing, or present symptoms of complications, a surgical solution should be considered. In larger ones (more than 10 cm), treatment must be immediate surgery.

Solid ovarian tumors are more frequently malignant, especially when they occur in women over 40 years of age.

Functioning tumors will require endocrine evaluation for a more accurate diagnosis.

It should not be forgotten that large or rapidly growing tumors at the extreme ages of life have great malignant oncological significance (children, adolescents and menopausal women).

Finally, the patient may present with an acute abdomen or the same may occur in a patient who has a previous diagnosis of ovarian tumor.

The most frequent complications of ovarian tumors are: torsion, hemorrhage, rupture, infection and malignancy of a benign neoplasm.

ENDOCRINOMETABOLIC SYSTEM. REGULATION NEUROENDOCRINE AND MAIN SYMPTOMS

71

NEUROENDOCRINE REGULATION

The endocrine system provides integrative or modulating mechanisms that allow the mammalian organism to adapt to a continually changing environment. This usually occurs with the nervous system, which works closely coupled to the previous one. Nowadays it is not logical to think of a nervous system and an endocrine system, without an integration between the two.

The hypothalamic-pituitary unit is very close and interrelated, so at the present time they cannot be separated, since they constitute an intimate and functional whole.

The hypothalamus plays a regulatory role in pituitary function through neurohormones, which are generally neuropeptides. The hypothalamus, through the portal-pituitary venous system, modulates the functions of the anterior pituitary through neurohormones called releasing hormones and exerts control over pituitary hormones. However, the neurohypophysis itself is a direct extension of the nervous system and functions as a reservoir for the axons of the supraoptic and paraventricular nuclei, producers of the hormones oxytocin and vasopressin, respectively.

Another level of integration of the nervous system with the endocrine system is represented by changes in volume and osmolarity, which determine variations in the secretion of hormones, such as aldosterone in the adrenal cortex and vasopressin in the hypothalamus (in the paraventricular nucleus) and released in the neurohypophysis.

It is undeniable that, like the nervous system, the endocrine system plays an adaptive and vegetative role.

Pituitary growth or somatotropic hormone (TSH), corticotropic hormone (ACTH), cortisol, aldosterone and antidiuretic hormone (ADH) modify the serum concentration of Nat. But in each of these groupings it is difficult to know, in given circumstances, which hormone is quantitatively more important and to calculate precisely how the various hormones, with their synergistic or opposing supplementary actions, work together to regulate the serum concentration of glucose or Nat. Furthermore, it is difficult to verify how the secretion of adrenaline by the adrenal medulla is related to the secretion of thyroid hormone; How the secretion of growth hormone is related to the secretion of thyroxine, and how aldosterone and gonadotropins have interrelationships. There are at least as many examples of independent functions as there are interrelated functions in the endocrine glands. Therefore, it seems more appropriate not to consider the endocrine glands, a system as closely related as the nervous system, but rather a group of secretory organs, of different embryological origins, that have appeared at different stages of evolution and that have a great diversity of functions, some separate and distinct, others interdependent. These regulatory functions are exerted by circulating chemical substances that act on different processes in cells and tissues.

Disorders of the endocrine glands are caused by excess, decrease or inadequate secretion of a hormone; either due to primary disease of the endocrine gland or due to abnormal secretion, in response to disease of some other organ (see below the section "Categories of endocrine diseases").

All endocrine diseases have this property in common: hormone secretion is not regulated, or is inadequately regulated, by control mechanisms that act under normal conditions; that is, hormone production is autonomous, anarchic or inadequate, a situation that causes a disorder of homeostasis.

NATURE OF A HORMONE

A hormone is a chemical substance secreted by a cell, or a group of cells, that exerts a physiological effect on other cells in the economy.

Some hormones are local, such as acetylcholine, which is released in parasympathetic nerve endings and skeletal muscle. General hormones are secreted by specific endocrine glands and transported by the blood to cause physiological actions at distant points in the economy. Some, like growth hormone, affect all, or almost all, cells in the body; others, such as corticotropin, have a specific effect on the adrenal cortex.

The tissues affected in this way are called target or white tissues. In future chapters we will present some examples of white organs or targets.

The different general hormones that have been shown to be of fundamental importance are the following:

1. Hormones of the anterior pituitary gland.
2. Posterior pituitary hormones.
3. Adrenal corticosteroid and adrenal medulla hormones.
4. Thyroid hormones.
5. Pancreatic hormones.
6. Ovarian hormones.
7. Testicular hormones.
8. Parathyroid hormones.
9. Placental hormones.

RETROCONTROL OR FEEDBACK OF HORMONAL SECRETION

When a hormone has begun to exert its physiological function, the intensity with which it is secreted begins to decrease. This depends on negative feedback, an important fact in the nervous control system. When it achieves the normal physiological effect, the secretion information reaches the producing organ in some form; If the gland secretes little, the physiological effects of the hormone decrease, in turn, the feedback decreases, by virtue of which the gland begins to secrete normal amounts of hormone again. In this way, the intensity of hormonal secretion is controlled, according to needs.

This mechanism is what has been called feed back, translated into Spanish as feedback in many books, although the term retrocontrol seems better to us.
In general, the feedback is negative, that is, the peripheral hormone exerts an inhibitory action on the hypothalamus.

There are positive feedback controls, such as that exerted by estrogen in the middle of the ovarian cycle, whose progressively elevated values determine a maximum elevation or peak of luteinizing hormone (LH), which allows ovulation.

Another example would be triiodothyronine (T.), whose elevation causes an increase in thyrotropin-releasing hormone (TRH).

CHEMISTRY OF HORMONES

There are basically two chemical types of hormones:

1. Hormones formed by proteins, protein derivatives or amino acids.
2. Steroid type hormones.

In the case of the glandular pituitary gland and pancreas, hormones are polypeptides or proteins.

The hormones of the pituitary gland, thyroid and adrenal medulla are made up of protein derivatives or amino acids.

Steroids are secreted by the supra-renal cortex, the ovaries and the testes.

Each hormone binds for its effect to specific receptors for each of them, which are synthesized inside the cell dynamically and in response to the needs of the human organism. The receivers for

Thyroid and steroid hormones are intracellular proteins that take up hydrophobic ligands as well as DNA.

Another family of membrane receptors are those that interact with the protein on the cell membrane.
This is the basic mechanism of glycoprotein hormones that have a higher molecular weight.

INTRACELLULAR HORMONAL MEDIATORS

Many hormones exert their effects on cells through the initial formation, in said cells, of 3'5' cyclic adenosine monophosphate (cAMP). cAMP gives rise to hormonal effects in cells. It is an intracellular hormonal mediator that has been called the second messenger by Sutherland (fig. 71.1).

It is thought that the stimulating hormone (first messenger) acts at the level of the target cell membrane, perhaps combining with a specific receptor for this type of hormone. The specificity of the receptors establishes which hormones can act on the target cells.

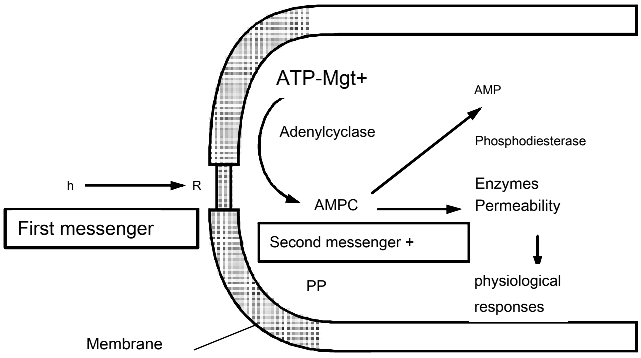


Fig. 71.1 Model of the second messenger: R, hormone receptor on the external side of the membrane. Phosphodiesterase can be inhibited by xanthine derivatives.

After the hormone is fixed on the receptor, the enzyme adenyl cyclase is activated in the membrane; The part of it in contact with the cytoplasm immediately produces the transformation of cytoplasmic adenosine triphosphate (ATP) into AMPe. cAMP then triggers a series of cellular functions before being destroyed: increase in enzymes in the cell, modification of cell permeability, etc., beginning the synthesis of specific substances. For example, a thyroid cell, under the stimulation of cAMP, forms thyroid hormones, while a cell of the adrenal cortex produces the synthesis of adrenal corticohormones.

Antidiuretic hormone (ADH) acts as a first messenger (extracellular), which activates adenyl cyclase, which acts as a second messenger (intracellular), which produces an increase in cAMP.

By acting on the epithelial cells of the renal tubule, cAMP increases their permeability to water.

cAMP is the intracellular mediator of at least the following hormones: thyrotropin, corticotropin, melanocyte-stimulating hormone, antidiuretic hormone, glucocorticoids, thyroid hormone, luteinizing hormone, insulin, glucagon, parathyroid hormone, catecholamines, serotonin and estrogens.

It has been thought that other intracellular mediators such as prostaglandins may play a similar role. These cyclic fatty acids were originally discovered in the seminal fluid. Their action, in addition to affecting the central nervous system and the digestive, cardiovascular and renal systems, has many effects on the endocrine organs: they affect the hypothalamus-anterior pituitary axis so that it increases the in vitro secretion of growth hormone; they allow the effects of luteinizing hormone on the ovary; cause lysis of the yellow body; alter the thyroid reaction to thyrotropic hormone (TSH): and affect steroidogenesis in the adrenal cortex. They mimic the hormonal effects of cAMP, and, in certain conditions, attenuate its effects. Although these effects of prostaglandins often appear to be hormonal, it has not yet been agreed to consider them as such.

MECHANISMS INVOLVED IN THE DIFFERENT CATEGORIES OF ENDOCRINE DISEASES

In the clinic the following classification may be useful:

1. Hyperfunctions, which can be:
    - a) Primary, due to increased hormonal secretion of the specific gland.
    - b) Secondary, of pituitary origin.
    - c) Tertiary, of hypothalamic origin.
2. Deficiencies, which can also be:
    - a) Primary (of the gland).
    - b) Secondary (of the pituitary gland).
    - c) Tertiary (hypothalamus).
3. Functional disorders of the endocrine glands.
4. Insufficiency of response of an end organ to some hormone.
5. Production of an abnormal hormone by an endocrine gland.
6. Production of hormones by a non-endocrine organ.
7. Iatrogenic endocrine disease.

PRIMARY HYPERFUNCTION OF ENDOCRINE GLANDS

In most cases it is due to benign tumors, such as functional pituitary, adrenal and thyroid adenomas.

Malignant tumors that secrete hormones are rare; We can find them in the pituitary gland, thyroid and the islets of Langerhans (insulinomas), among others.

There are states of hyperfunction that are not due to endocrine tumors, such as Basedov's disease or diffuse toxic goiter.

SECONDARY HYPERFUNCTION OF THE ENDOCRINE GLANDS

It defines the excessive activity of a target organ due to hyperfunction of the anterior pituitary gland; For example, Cushing's syndrome produced by excessive and persistent secretion of ACTH, without adrenal tumor.

ENDOCRINE GLAND INSUFFICIENCY

Endocrine gland insufficiency can be primary, secondary and tertiary.

As examples in the thyroid gland, primary insufficiency would be autoimmune hypothyroidism, due to primary damage to the thyroid gland; the secondary one, the hypothyroidism that is observed in the pituitary lesion, which causes Sheehan's syndrome and the tertiary hypothyroidism, if it occurs as a consequence of a hypothalamic lesion.

Primary insufficiency is the most common cause of endocrine insufficiency syndromes.

"FUNCTIONAL" DISORDERS OF THE ENDOCRINE GLANDS

The response of the endocrine glands to certain pathological states may or may not be important.

Examples of responses that do not have proven clinical significance are presented in: modification of thyroxine metabolism in liver diseases, acute disorders due to various causes, altered cortisol metabolism in portal cirrhosis, altered androgen metabolism in thyroid diseases, acute intermittent porphyria, etc.

On the contrary, secondary hyperparathyroidism occurs in renal failure, which is the cause of the so-called renal osteodystrophy.

INSUFFICIENCY OF RESPONSE OF A TERMINAL ORGAN TO ANY HORMONE

In nephrogenic diabetes insipidus there is an absence of response due to a defect in cellular receptors to vasopressin or antidiuretics (ADH).

Something similar happens with pseudohypoparathyroidism where there is an absence of bone and kidney response to parathyroid hormone (PTH).

Testicular feminization syndrome is another example of a target organ defect, due to a defect in testosterone receptors and virilization does not occur.

PRODUCTION OF AN ABNORMAL HORMONE BY AN ENDOCRINE GLAND

It is observed in inborn errors of metabolism:

- In congenital adrenal hyperplasia, the deficiency of hydroxylases in C21 and C1l causes metabolic blockage in the synthesis of cortisol; The result is that various precursors such as pregnantriol and 17-0-hydroxyprogesterone, which normally exist in very small quantities, are produced in much larger quantities, as are androgens at C19.

- In cretinism with goiter, the blockage in thyroxine synthesis results in an increase in thyroid secretion that sends large amounts of monoiodotyrosine and diiodotyrosine into the blood.

In so-called "T" (triiodothyronine) thyrotoxicosis, there is hyperthyroidism, but protein-bound iodine concentrations and serum thyroxine concentrations are normal. For unknown reasons, the larger gland or thyroid nodule synthesizes excessive amounts of triiodothyronine (T,), which is normally less abundant.

PRODUCTION OF HORMONES BY A NON-ENDOCRINE

Cancers of various organs: lung, thymus, pancreas, among others, can produce substances that cannot be distinguished chemically or immunologically from normal endocrine products, generally proteins such as ACTH, gonadotropins, erythropoietin, melanotropic peptides and other hormones.

Nas.

It is not absolutely proven that tumors release and secrete hormones, but it is almost certain; However, these secreted substances need to meet certain requirements for this fact to be accepted. These requirements are:

1. Coexistence of tumor and endocrine syndrome.
2. Demonstration in blood or urine of the causative hormone.
3. Demonstration of large amounts of the hormone in the tumor and that it is not coming from the gland that normally produces it.
4. In vitro demonstration of the tumor's ability to produce large amounts of hormone (would be ideal).
5. Disappearance of the syndrome or improvement after removing the tumor.

IATROGENIC END0CRINE DISEASE

Iatrogenic Cushing's syndrome due to the administration of ACTH, cortisol and their derivatives is the most common.

Another example is the prescription of sex hormones and thyroid hormone to patients without gonadal or thyroid deficiency, which can lead to endocrine syndromes. Methyltestosterone can cause cholestatic jaundice; contraceptives can cause metabolic abnormalities.

Protein hormones sometimes cause discomfort. There are published cases of anaphylaxis caused by the administration of ACTH.

ROLE OF THE HYPOTHALAMUS IN ENDOCRINE REGULATION

The hypothalamic-antepituitary axis includes antepituitary hormones and neurohormones from the hypothalamus. These substances are: CRH (corticotropin-releasing hormone), GHRH (somatotropin-releasing hormone), TRH (thyrotropin-releasing hormone), LHRH (luteinizing hormone-releasing hormone) and FSHRH (follicle-stimulating hormone-releasing hormone). Hypophysiotropic inhibitory hormones have also been identified: they are PIF (prolactin inhibitory factor) and GHIH (growth hormone inhibitory hormone or somatostatin). The secretion of melanotropic hormone (MSH) by cells of the intermediate lobe in animals, which do not form a defined anatomical subdivision in humans, is not proven in humans, but the existence of melanocytostimulating peptides is and the melanocytostimulating effect of ACTH has also been proven.

These hormones control the secretion of the different endocrine glands subject to the pituitary gland (thyroid, gonads, adrenals) and intervene in all metabolisms (protein, carbohydrate, lipid and hydromineral). They play an essential role in growth, maintaining general metabolic activity and reproduction.

Hypothalamic neurohormones are probably proteinaceous in nature; Each of them acts specifically on one of the pituitary hormones and can be called hypophysiotropic. Some have a stimulating role (GH-releasing hormones, ACTH, follicle-stimulating hormone FSH, and LH);

others, an inhibitory role (hormones that inhibit prolactin and melanocytostimulating peptides). The TSHo-releasing hormone TRH has been isolated and synthesized, as has the decapeptide that stimulates the secretion of FSH and LH: FSH-releasing hormone-LH and the melanocytostimulating peptide inhibitory factor (MIF).

The exact place where neurohormones are secreted has not yet been determined, although the hypothalamic areas where some of them originate are known. From their origin, the hormones travel through the axons of the secretory neurons that end in the median eminence. At this level, neurohormones are released in the primary capillaries of the infundibuloantepituitary portal system, which transports them to the anterior lobe where they can act.

The level of secretion of hormones from the hypothalamic-pituitary axis is subject to two types of control: nervous and humoral. Nervous control acts essentially on hypothalamic neurohormones and, through their mediation, on antepituitary secretions. In addition, it involves numerous sensory, neuropsychic and physicochemical stimuli.

Humoral control consists of a series of regulations between the different hypothalamic and pituitary hormones and the endocrine glands eventually placed under their dependence. Each of the hormones produced by these glands generally acts by long negative feedback on the corresponding neurohormone. But each one also has a direct action on the pituitary hormone, as has recently been demonstrated for thyroxine, estradiol and synthetic corticosteroids. Experimentally, a positive retrocontrol has been observed in the rat for the LH-releasing hormone.

There are other forms of control: interrelationships between hormones from peripheral glands (thyroid, adrenal, gonads) and internal or short feedback, in which the pituitary hormone could act on the corresponding neurohormone through negative feedback.

The hypothalamic-postpituitary axis includes two so-called postpituitary hormones: oxytocin and vasopressin. These two nonapeptides are made by hypothalamic neurons (supraoptic and paraventricular nuclei) and are also neurohormones. They are stored in the posterior lobe of the pituitary gland at the end of the axons of neurosecretory cells. They are released by the action of various stimuli. Oxytocin is specific for the contraction of the smooth fibers of the uterus and the myoepithelial cells of the mammary gland, while vasopressin acts on the smooth fibers of the vessels (vasopressor action) and, above all, on the facultative absorption of water at the level of the Bellini tubules of the kidney (antidiuretic action).

The regulation of these hormones seems simpler than that of the hypothalamic-antepituitary axis. Stimulating hormones are generally excitations of the peripheral nervous system for Oxytocin (mammary or genital sensory receptors) or variations in osmolarity, pressure or blood volume for antidiuretic hormone.

CONTROL OF BIOGENEIC AMINES ON HYPOPHISOTROPIC FUNCTIONS

The hypophysiotropic neurons are in turn innervated by monoamine neurotransmitter neurons (Fig. 71.2), of which the main ones secrete dopamine, norepinephrine and serotonin. It seems likely, although it has not been proven, that norepinephrine stimulates the secretion of LHRH, TRH and GHRH, and dopamine stimulates the secretion of PIF. There is some data that serotonin possibly inhibits the secretion of PIF and TRH.

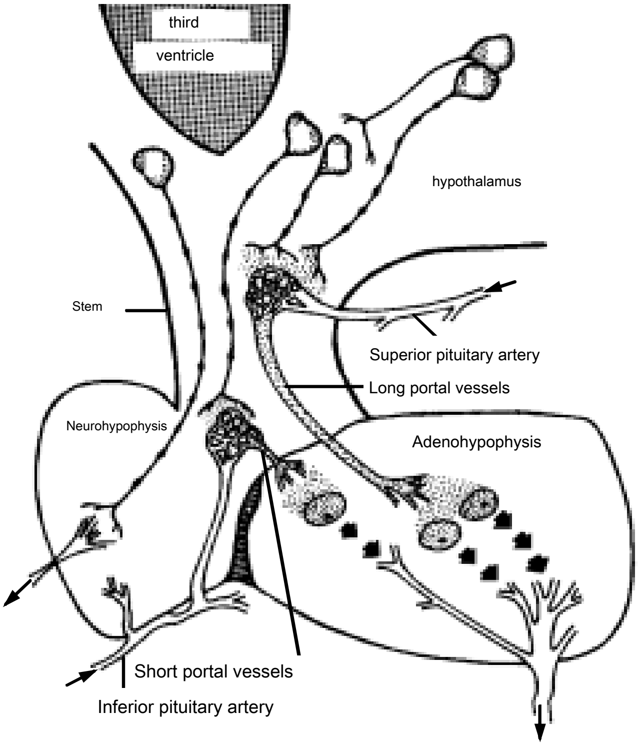


Fig. 71.2 Functional elements of the control of the hypothalamic-pituitary unit. Two general types of neurons participate in the regulation of the anterior pituitary. The first type, the peptidergic neurons, form the releasing hormones and end in the median eminence (1) or in the pituitary stalk (2), related to the capillary plexus of the portapituitary vessels: they are also called tuberohypophyseal neurons; They are neurosecretory, that is, they combine the functions of being an electroexcitable tissue and having a secretory function; They act as neuroendocrine transducers (Wurtman, 1971), to transform neural information into hormonal information, with a function similar to that of the classic supraoptic-pituitary neuron (3), which is responsible for the secretion of vasopressin. The second type of neuron corresponds largely to the so-called monoaminergic neurons;

They constitute the junction between the rest of the brain and the peptidergic neurons and it is believed that they end on their cell body, in the ordinary form (4) or at the end of their axons (5) (axoaxonic form, by Schneidery McCann).

MAIN SYMPTOMS OF THE SYSTEM ENDOCRINOMETABOLIC

We are going to study here only the main symptoms for which patients consult and that may be related to alterations of the endocrine-metabolic system.

These symptoms are:

-Asthenia.

- Polyuria.

-Polydipsia.

- Polyphagy.

Obesity.

-Thinness.

- Increased thyroid volume (goiter).

- Exophthalmia or exophthalmos.

-Size alterations.

- Hirsutism.

ASTHENIA

Concept

According to its etymology, asthenia is the loss of energy or the total absence of strength with a feeling of exhaustion or tiredness. Its meaning is different from that of fatigue, which is a physiological state.

Patients refer to it as weakness, tiredness, exhaustion, lack of energy, etc. There is an inability to do what they normally did before or what others do easily.

semiography

Asthenia can be mild, moderate or severe.

Light. It is observed at the beginning of certain metabolic, endocrine, infectious and even neoplastic diseases.

Moderate. Like the one that occurs in the period of state of the indicated conditions and that still allows some physical activity.

Intense. Here the asthenia is so severe that it draws attention due to the degree of disability it produces. The patient can barely lift an arm, open his eyes, or swallow.
As examples we have asthenia in myasthenia gravis, Addison' s disease or the final periods of cancerous diseases, acute hepatitis, etc.

The degree of asthenia does not always denote the severity of the condition that produces it; There are patients with very serious conditions who do not show asthenia and perhaps are carriers of a neoplasia, while others who feel extraordinarily exhausted are carriers only of psychoneurotic alterations.

Semidiagnosis

Asthenia is observed in:

1. Brain processes. In regulation disorders due to organic or functional alterations of the reticular formation and hypothalamic centers.
   It is observed above all in psychoneurotics in the form of nervous fatigue, which rather than fatigue is an asthenia upon awakening, which subsides during the course of the day.
   It is more common in people with an asthenic habit.
   It also accompanies school and sports supplements, and neuropathies (psychosomatic asthenia).
2. Anemias. In these cases, asthenia is caused by hypoxia.
3. Metabolism disorders. Asthenia is typical of hyperlipidemic and acidotic patients.
4. Electrolyte imbalances. Asthenia is due to hyponatremia and hypokalemia.
5. Endocrinopathies. Asthenia is common in endocrine insufficiencies. For example, hypothyroidism (thyroid asthenia), adrenocortical insufficiency (Addison's disease), gonadal insufficiency (asthenia of menopause and andropause), Cushing's disease, pituitary insufficiency, and hypoglycemia (hypoglycemic fatigue).
6. Infections. Mainly in tuberculosis.
7. Malignant neoplastic processes.
8. Permanent arterial hypotension.
9. Myasthenia gravis.
10. Senile age.

Of all these causes, cancer, tuberculosis and psychological disorders are the most important and those that should not be forgotten. The association of asthenia, melanoderma and arterial hypotension constitutes the clinical triad of chronic adrenal insufficiency.

POLYURIA

It was studied in Chapter 66, corresponding to the urinary system.

POLYDPYSIA

It presents as an excessive sensation of thirst that forces the patient to drink large amounts of water, sometimes 15 times the most. It generally accompanies polyuria, giving rise to the polyuric-polydipsic syndrome.

Semiogenesis and semidiagnosis

Below we point out the most common polydipsias, as well as their mechanisms and causes:

1. Psychogenic polydipsia or potomania.
2. Polydipsia due to alteration of thirst regulatory mechanisms.
3. Polydipsias veras (retrohypophyseal):
   a) Hereditary.
   b) Idiopathic itself.
   c) Secondary to pathologies: mumps, histiocytosis X, erythroleukemia, etc.
   d) Secondary to iatrogenic injuries (surgical, radiophysical).
4. Nephrogenic polydipsias:
   a) Congenital.
   b) Isolated.
   c) Associated with tubulopathy (Toni-Debré-Fanconi syndrome). This syndrome is rare and only occurs in childhood. It is due to the insensitivity of the renal tubules to the antidiuretic effect of vasopressin; It is, therefore, resistant to pitresin. Sometimes it is hereditary.
5. Hypokalemic polydipsia. It causes tubular lesions that prevent water reabsorption. It is accompanied by K depletion due to:
   a) Primary hyperaldosteronism.
   b) Cushing syndrome (not in all cases).
   c) Overdosage of desOxicorticosterone.
   d) Intense diarrhea.
6. Hypercalcemic polydipsia (plasma Ca greater than 11 mg/ 100 mL). Due to hyposthenuria secondary to nephrocalcinosis and a possible Ca inhibitory effect:
   a) Excessive mobilization of bone tissue: hyperparathyroidism, bone metastasis, fractures, resting osteoporosis, long-term metabolic acidosis, etc.
   b) Excessive reabsorption of Ca in the digestive tract: vitamin D poisoning, digestive sarcoids, milk-alkali syndrome (Burnett syndrome).
   c) Idiopathic hypercalcemia (only in children, very rare): Lightwood and Payne syndrome, hypersensitivity to vitamin D, vitamin D overdose, Fanconi syndrome (idiopathic infantile hypercalcemia), García syndrome (with osteosclerosis and congenital malformations), etc.

POLYPHAGY

It consists of the exaggeration of the desire to eat, and is characterized by a voracious hunger for any food.

Semidiagnosis

It is presented in:

1. Gastroduodenal ulcer. The patient eats to relieve the discomfort and pain caused by hypersecretion with hyperchlorhydria.
2. Diabetes. There is true polyphagia, generally associated with polyuria and polydipsia.
3. Convalescence. Especially in infectious diseases.
4. Intestinal parasitism (due to tapeworm, etc.).
5. Psychoneurosis. Polyphagia exists as a compensation for the decrease in sexual instinct, according to some authors.

bulls.

6. Mental illness.
7. Hysteria.
8. Mongolism.
9. Pregnancy. There is capricious appetite (cravings) quite frequently.

OBESITY

Synonymy

It is also known as adiposity, pimelosis and adipose polysarcia.

Concept

It is the increase in adipose tissue due to hypertrophy or hyperplasia of fat cells.

Any patient who is more than 20% above the ideal weight is considered obese.

The obese person is a corpulent individual, exaggeratedly fat. In it, energy is excessively accumulated or stored in adipose tissue and produces morphological, nutritional and metabolic changes.

Obesity is one of the metabolic disorders that has been known since ancient times. However, its frequency is unknown due to the difficulty in measuring the degree of obesity. It is not adequately reported in hospital and outpatient statistics.

30% of men and 40% of women are 20 pounds or more over their ideal weight (overweight).

This overweight is more common in men between 40-49 years of age and in women after 50 years of age, and is associated with a sedentary life (little physical activity).

Hypernutrition is the most common form of malnutrition in developed countries, and obesity is the most common faithful exponent. Let us remember the words of a clinician: "they dig the grave ahead of time with their teeth."

Obesity maintains a constant increase in developed countries, and in Cuba the practice indicates a similar fact. Its incidence is more or less than 30%. It is found three or four times more frequently associated with diabetes mellitus.

Obesity has been associated with peripheral insulin resistance, which causes hyperinsulinism, hirsutism or increased body hair in women, high blood pressure, hyperlipoproteinemia (elevated plasma lipids) and, finally, leads to atherosclerosis. This set of vascular risk factors is called syndrome X. These symptoms and signs present a common production or pathophysiological mechanism, due to insulin resistance.

Semiogenesis or pathophysiology

Obesity is a syndrome of multiple etiologies, but with similar production mechanisms. In volume 1, Chapter 24, general reference is made to the factors involved in its production, which will now be studied in detail.

From excessive food intake or a decrease in energy expenditure, hypertrophy or hyperplasia of adipose tissue occurs, causing a decrease in insulin receptors and a decrease in glucose tolerance, expressed by hyperglycemia and hyperinsulinism, which leads to an increase in the synthesis of very low-density lipoproteins (VLDL), whose function is to transport endogenous triglycerides, which causes atherosclerosis.

Obesity is called symptomatic or idiopathic, depending on whether it is the expression of an individualizable disorder or not. The idiopathic obesity group is the largest and poses very complex and still unresolved problems regarding its etiopathogenesis and pathophysiology.

In general terms, it is considered that two types of hormones are involved in the production of obesity;
the endogenous and the exogenous, which frequently add up in variable proportions in the same subject.

Endogenous factors

The most important endogenous factors, according to experimentation on animals and humans, are:

- Neurological factors.
- Psychological factors.
- Endocrinometabolic factors.

Neurological factors (fig. 71.3)

In-depth studies of histophysiology and electrophysiology have made it possible to ensure that in the hypothalamus there are areas that govern food intake. Of them

A ventrolateral one has been described, in which the so-called appetite center is located, whose stimulation conditions the appearance of bulimia and its destruction determines anorexia. This center receives stimuli from the ventromedial area, where the satiety or regulatory center is located, which inhibits the previous one and sends information to the cerebral cortex, to govern the complex sensation of appetite. When this center is destroyed, uncontrollable hyperphagia appears.

There has been much discussion about what stimulates this ventromedial or satiety center in its action on the appetite center; It is thought that the most probable thing is that the decrease in the difference between arterial and venous blood glucose inhibits the activity of the satiety center and determines the excitation of the appetite center, while the increase in this difference after food intake would stimulate it, with the consequent opposite result. It is the mechanism that called glucostat. The previous mechanisms of the thermostat (thermal differences) and the lipostat (variations in the speed of lipid mobilization) have been discarded.

Gastric distension occurring after food intake would cause stimuli that would reach the hypothalamus and excite the satiety center, which would inhibit the appetite center. On the contrary, if the appetite center were released, gastric contractions would occur that would contribute to creating the unpleasant sensation of hunger, which signals the urgent need to eat food.

In the human species, the percentage of obese people caused by a primitive anatomical lesion of the hypothalamus is very low; This injury is more frequently observed (secondary) due to craniopharyngiomas, meningitis, trauma and post-surgical sequelae of the skull base.

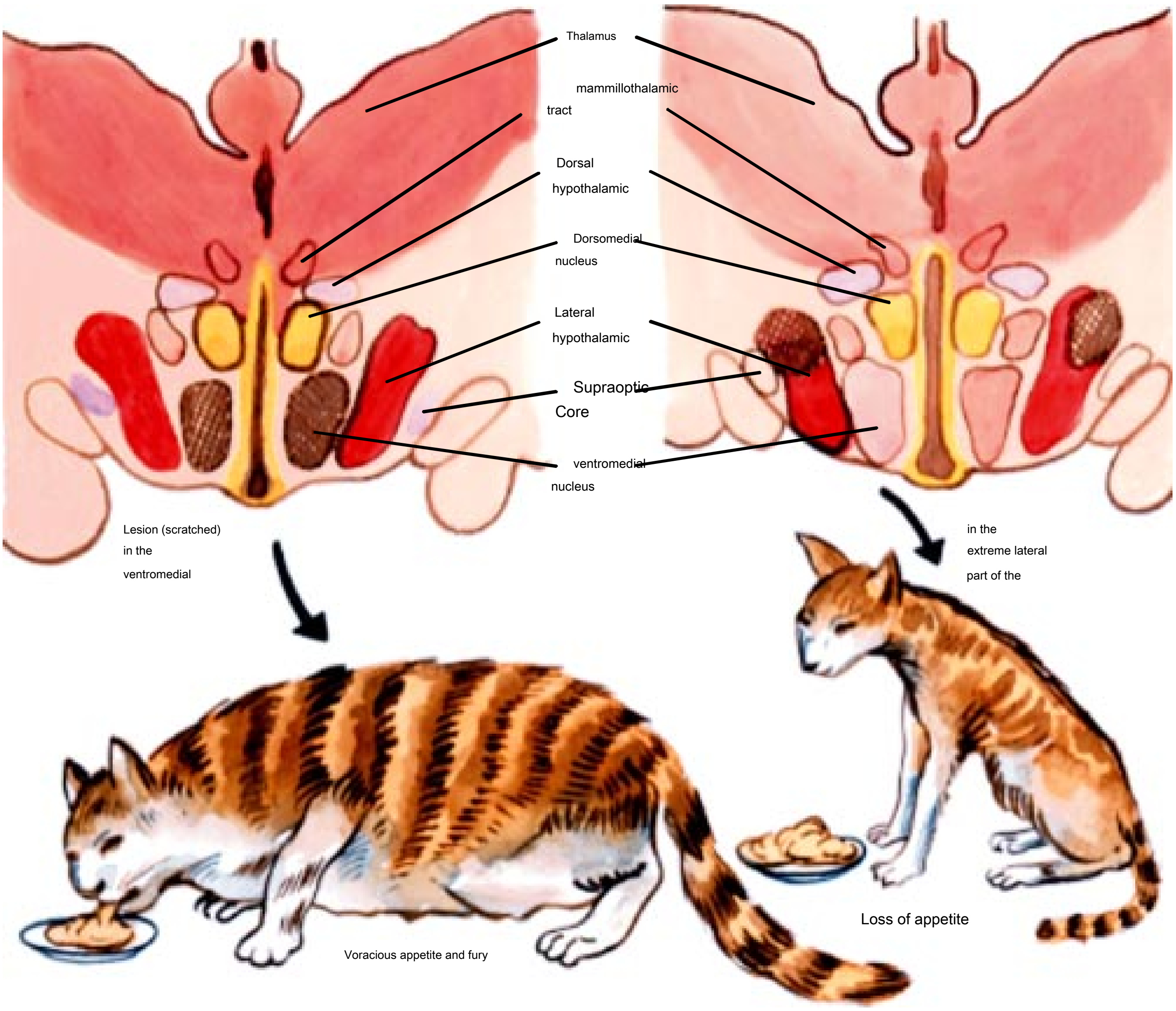

Thalamus
mammillothalamic tract
Dorsal hypothalamic
Dorsomedial nucleus
Lateral hypothalamic
Supraoptic Core
ventromedial nucleus
Lesion (scratched) in the ventromedial
in the extreme lateral part of the
Loss of appetite
Voracious appetite and fury

Fig. 71.3 Center for the control of satiety and appetite.

Psychological factors

They are the most difficult to value; They include anxiety, dissatisfaction, emotional conflicts and others.

It has not been proven whether they exert their influence through the hypothalamus.

Endocrinemetabolic factors

Through precise metabolic studies it has been possible to experimentally verify, both in animals and in the human species, that in a percentage of obese people there are metabolic disorders of carbohydrates and water and saline exchange, which explain the ease with which obesity occurs in these subjects and also that they are refractory to the usual therapeutic measures. It is particularly interesting to note that these alterations can be permanent, as seems to happen in some individuals for stable genetic reasons, or transitory, reversible or modifiable during treatment.

With regard to lipid metabolism, two fundamental alterations have been verified in adipose tissue: the decrease in lipolysis, possibly due to a lower activity of an intracellular lipase, which is demonstrated by the lower increase in circulating free fatty acids, as a consequence of fasting or the supply of some of the major stimulants of lipolysis (growth hormone, catecholamines or triiodothyronine), and the increased lipogenesis, which determines greater biosynthesis of lipids, especially from carbohydrates, as a consequence of an enzymatic alteration that favors this condition, which is not precise in nature, but is linked to an increase in insulinemia.

The algebraic sum of these two factors: decreased lipolysis and increased lipogenesis, based on modifiable metabolic alterations in a significant percentage of these patients, is what conditions, maintains and makes it difficult for them to reduce the accumulation of adipose tissue.

A third type of metabolic disorder, particularly important to explain certain difficulties in achieving adequate weight reductions, is related to water and salt metabolism. It has been shown that in obese people there is a decrease in glomerular filtration of water and sodium, which becomes greater with the individual standing, and there is also an increase in antidiuretic hormone and aldosterone that is accentuated with massive carbohydrate intake. During reduction treatments, these metabolic anomalies are accentuated by the production of water that is generated, as a consequence of lipolysis, which reaches at least 1 mL/g of adipose tissue that is catabolized.

In a small percentage of cases these metabolic disorders are permanent and of genetic origin, while in the vast majority of individuals in whom they have been verified, they generally appear after a more or less prolonged time in which the subject has become progressively more obese. The reason for this is unknown, but in experimental animals it has been shown that hepatic enzymatic activity and lipogenetics increase extraordinarily after the accumulation of hepatic triglycerides has exceeded a certain level.

In summary, in all obese people, the abnormal accumulation of adipose tissue is determined by excessive caloric intake with respect to consumption. In a small percentage of these patients, certain metabolic alterations of carbohydrates and lipids favor this abnormal deposit and subsequently make its reduction difficult. These alterations are unknown, but they are related to a large prior accumulation of lipids, with the development of adipose cells. Exceptionally, these alterations are genetic. That is to say, in almost all obesity a dynamic phase occurs, determined by the simple energy imbalance between intake and consumption, which is followed by a metabolic phase that accentuates (reducing consumption) the consequences of the altered balance.

To reaffirm the above, it is enough to point out that up to 25 altered parameters have been identified in this type of metabolically obese person, among which a non-hypothyroid hypometabolic state deserves to be highlighted. Particularly in this type of obese person, the distribution of the same caloric intake is of utmost importance, since experimental studies have shown that a single massive intake causes the appearance of a much larger amount of adipose tissue than when the same amount of food is supplied subdivided several times a day.

Exogenous factors

Regarding exogenous factors, they acquire a special significance in moderate forms of obesity, and therefore they must be kept in mind to institute complementary measures in the treatment of patients.

Within this category of hormones, the habits and customs of certain social groups must be considered, which tend to favor the intake of products with very high calorie content and the increasingly marked decrease in physical activity.

It should be known that within diets of similar caloric content, those richer in carbohydrates favor the greater accumulation of lipids and that, likewise, processes that determine more or less prolonged immobility can trigger obesity, sometimes truly monstrous.

semiography

ideal weight

According to Broca's formula, weight (in kg) is equal to height expressed in centimeters minus one hundred. For example, a man of 168 cm should weigh 68 kg, according to: weight = 168-100 = 68. This is a practical formula.

To know the ideal weight, you should always consult the existing tables (see tables 3.1, 3.2 and 3.3, in volume 1, Section I, Chapter 3). Some authors accept a normal deviation of 5% above the ideal weight;
others up to 10%.

It is considered:

Light obesity (mild).. 20-29% above the ideal weight.

Moderate obesity...30-39% above ideal weight.

Severe obesity. 40-99% above ideal weight.

Some call it monstrous, when it is almost deformed and of course, more so than those mentioned above (figs. 71.4 and 71.5).

Another measurement is the subscapular fold: a measurement greater than 22 mm in men and 27 mm in women is considered a criterion for

The measurement of the triceps fold is another indicator of obesity, when it is greater than 19 mm in men and greater than 30 mm in women.

At the present time, vital importance is given to the body mass index or Quetelet index:

$$\text{Body Mass Index} = \frac{\text{Body weight in kg}}{(\text{Size in m})^2}$$

This determination is diagnostic of obesity if it is greater than 27 in men and 25 in women.

Another important indicator is the waist-hip circumference ratio greater than 0.7 in men and 0.85 in women.

Clinical study

Obesity, in addition to being a syndrome, is a symptom and a sign. The capital symptom is overweight, which we determine from the existing tables. It is also necessary to calculate the degree of obesity. It is necessary to specify whether "fatness" is closely linked to adiposity, that is, if there is an excessive accumulation of fat, with a generalized distribution.

Not every increase in weight or volume is obesity.

Let us remember that there are very muscular individuals, individuals with a large skeletal framework and patients with edema. It is necessary to differentiate these cases from those

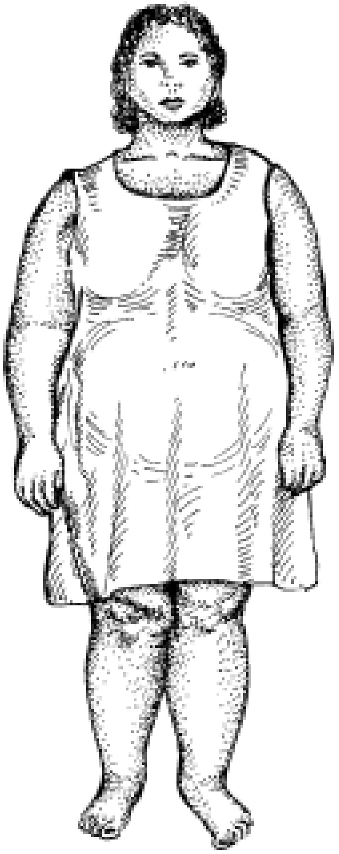
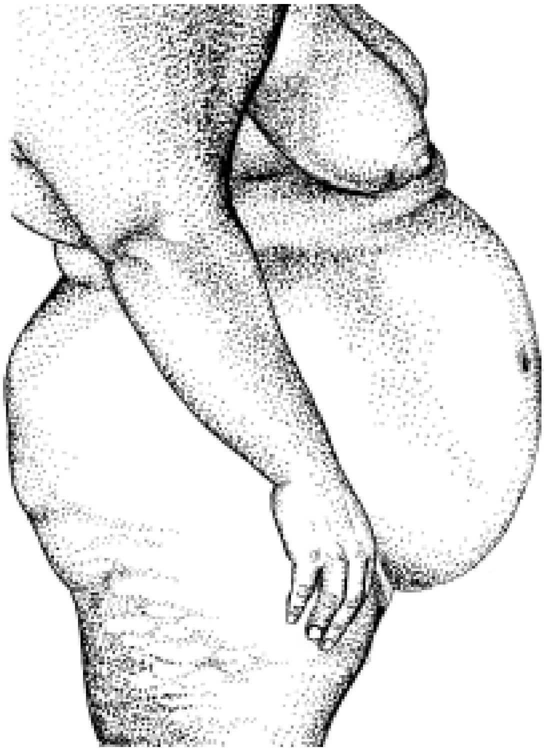


Fig. 71.4 Excessive obesity.

Fig. 71.5 Monstrous obesity.



that present regional accumulations (lipodystrophies) and circumscribed accumulations (lipomatosis).

Semidiagnosis

1. Exogenous obesity.
2. Endocrine obesity:
    a) Excess insulin or adrenocortical hormones associated with other endocrine disorders.
    b) Non-insulin-dependent diabetes mellitus.
3. Gonadal dysfunction:
    a) Ovarian: Stein-Leventhal syndrome.
    b) Male: Frölich syndrome.
4. Throid dysfunction. Hypothyroidism.
5. Cushing's syndrome.
6. Hypothalamic obesity. Traumas, tumors and inflammatory processes that take different clinical forms.
7. Pickwik syndrome; obesity syndrome with hypoventilation.
8. Genetic obesity. If it is from both parents, it appears in 80%.
9. Family obesity: Strong constitutional family incidence.
10. Obesity of undetermined origin. Also called essential or idiopathic.

Two types of obesity are also described from a morphogenetic point of view:

1. Gynoid obesity. Feminoid in nature, more common in women. Predominantly in the lower half of the body; It's purely adiposis.
2. Android obesity. Of the male type, it predominates in the upper half of the body and offers the sufferer greater activity and mobility than the previous one.
    This type of obesity is related to cardiovascular and vascular complications, which is why it represents an important vascular risk factor.

THINNESS

Concept

It is the state of the organism in which the weight is lower than its theoretical weight.

Even though the clinical terms of thinness or weight loss are used interchangeably, they are two concepts. In thinness, the individual has a weight lower than that corresponding to his or her height, age, and sex, without presenting any pathological process. Weight loss or malnutrition is an evolutionary and always pathological process, which ranges from the exaggerated use of reserves hydrocarbons and fats to the destruction of tissues. The extreme degree of malnutrition is called cachexia.

Although obesity and thinness seem to be two opposite poles, if we delve into their study we see that they are not completely antagonistic (see volume 1, Chapter 24).

semiography

The appearance of thin subjects is already well known. The fusion of adipose tissue and other tissues does not affect all regions in the same order and equally.
Each patient has their own way of losing weight. The fat on the belly, thighs and buttocks, as well as the fat on the face, disappears first. The eyeballs and cheeks are sunken, sucked in. Because the sinuosities of the facies are evident, these patients are attributed to be older than their age. The muscles of the neck, the Adam's apple, the clavicles, the ribs, the xiphoid process and the crests of the iliac bone form prominence. The belly is flat and excavated; dry arms.

There is a wide space between the adductor muscles of the thigh.

Semiogenesis and semidiagnosis

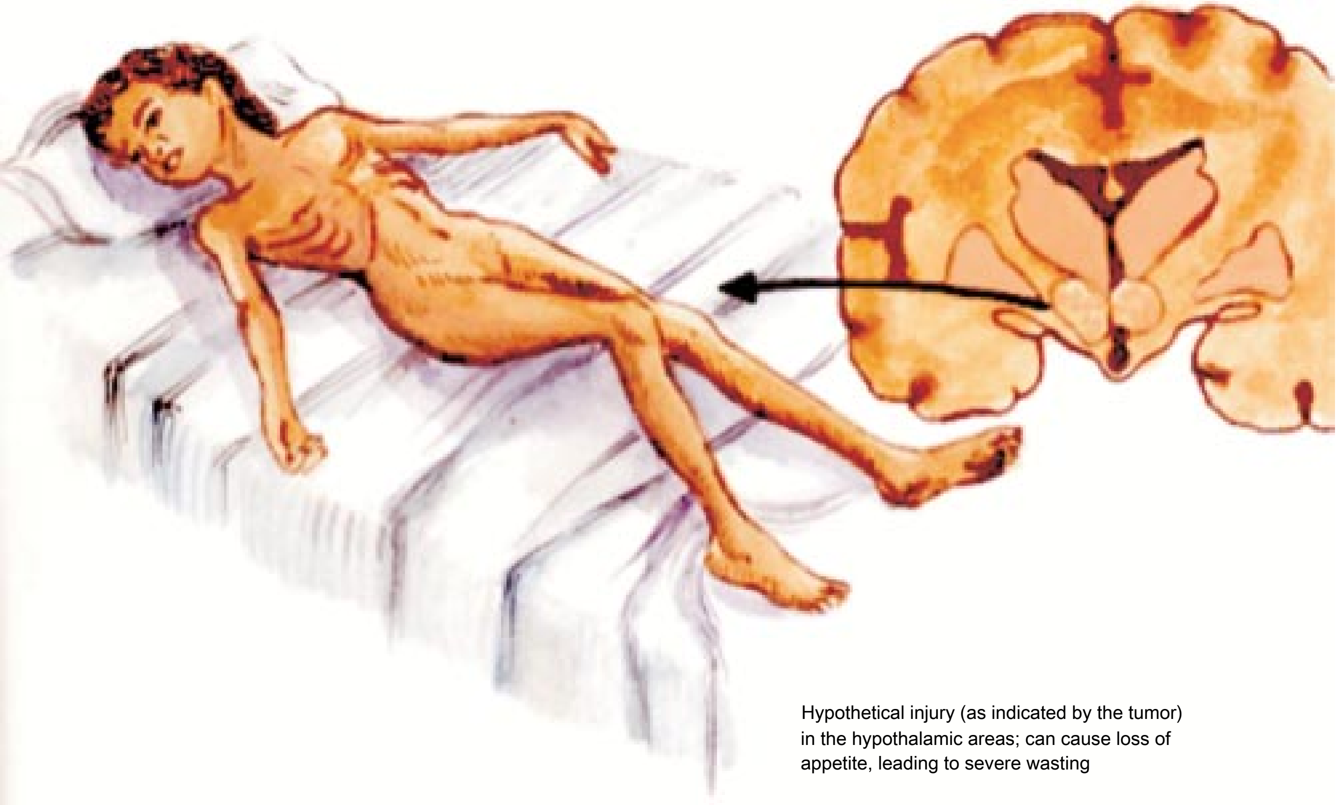
Thinness can be caused by: incorrect diet, illness and constitutional state.

Thinness due to incorrect diet

It is observed when the diet is quantitatively and qualitatively insufficient. We must not forget the paradoxical fact that due to a state of imbibition, edematous or preedematous (hunger edema) the individual's weight can increase.

The reasons for incorrect feeding may be:

1. Total or partial lack of food (absolute hunger or relative hunger, respectively).
2. Intense anorexia. It can be motivated by organic processes (neoplastic, infectious, febrile, uremic, endocrine) or psychological (endogenous depression, permanent emotional states, schizophrenia, etc.). Anorexia nervosa due to psychogenic or hypothalamic disorders deserves special mention (fig.71.6). Hamburger calls stigmatic anorexia the habitual lack of appetite in children, due to eating irregularly between main meals and due to the inconvenient composition of the diet.
3. Difficulty swallowing and swallowing (for example, missing teeth, esophageal cancer, etc.).



Hypothetical injury (as indicated by the tumor) in the hypothalamic areas; can cause loss of appetite, leading to severe wasting

Fig. 71.6 Anorexia nervosa due to hypothalamic damage.

Thinness due to illness

1. Endocrine. This type of thinness is characterized by an alteration of the basal metabolism or the intermediate metabolisms that are responsible for thinness.

Thyroid diseases can cause thinness, especially hyperthyroid states.

Simmonds cachexia and Sheehan syndrome, caused by lesions of the pituitary gland, have thinness as one of their most striking symptoms.

Chronic adrenal insufficiency or Addison's disease is accompanied by intense thinness.

2. By consumptive processes. These processes accompany chronic infections, neoplasms (especially of the digestive system), malignant hematological diseases such as leukosis and lymphomas, chronic tuberculosis, poisoning, etc.

This thinness is also typical of intestinal malabsorption syndromes, chronic diarrhea and parasitism, which are sometimes also associated with ingestion disorders and produce so-called mixed thinness.

Thinness by constitutional status

It is not a disease but a state. These are generally individuals with an asthenic habit: tall, long neck, drooping shoulders, winged scapulae, acute xiphoid angle, narrow thorax, pendulous belly and neurovegetative instability with slightly decreased or normal basal metabolism.

Many times they have a good appetite and, due to the amount of food they consume and do not gain weight, "it seems as if they were pouring the food into a bottomless bag."

Some authors describe constitutional thinness as essential or idiopathic.

Sometimes this thinness is familiar.

INCREASE IN THYROID VOLUME (GOITRE)

Concept

The increase in volume or abnormal thickening of the thyroid gland, regardless of its etiology, is called goiter.

Semiogenesis or pathophysiology

Goiter can be caused by three main types of alterations:

1. Functional hypertrophy and hyperplasia.

2. Inflammatory reactions.
3. Neoplasms.

Functional hypertrophy and hyperplasia

It may not cause a nodular goiter, but more frequently it produces diffuse or mixed goiters. Within this group, we have:

1. Non-toxic goiters. They are generally produced by a decrease in thyroxine that can be due to different factors (fig. 71.7):
    - a) Hypoiodism, due to absolute or relative lack of iodine uptake (trap): simple, endemic, iodopenic goiter, sometimes with hypothyroidism.
    - b) Expulsion of thioiodide, produced by thiocyanate and perchlorate.
    - c) Antithyroxinogenesis, produced by thioureas.
    - d) Defects in thyroid hormonegenesis, produced by genetic deficiencies of several enzymes.

    The common denominator in all non-toxic goiters is the deficiency of thyroid hormone and therefore, the marked increase in pituitary thyrotropic hormone, which is responsible for the enlargement of the thyroid gland.

2. Toxic goiter. It can be nodular (and in this case it is called Plummer's disease) or diffusely hyperplastic goiter, called Parry's disease, Graves' disease, Basedow's disease or, frequently, Graves-Basedow's disease. It is accompanied by excess thyroid hormones.

Inflammatory reactions

In these cases, goiter is due to the inflammatory response of the thyroid to various etiological agents;
constitute true thyroiditis:

1. Acute: not suppurative.
2. Subacute: granulomatous, de Quervain, etc.
3. Chronic: tuberculous, syphilitic, Riedel's thyroiditis, Hashimoto's thyroiditis, etc.

Neoplasms

Tumor development is responsible for the increase in volume. They can be:

1. Benign. They are adenomas (fetal microfollicular, macrofollicular, papillary cystadenoma).
2. Malignant. They are carcinomas (follicular, papillary, mixed, medullary and undifferentiated).

Some of these lesions are associated with states of euthyroidism, hypothyroidism or hyperthyroidism.

Semiotechnics

It is performed by inspection, palpation, measurement of the neck and auscultation (presence or absence of murmur in vascularized thyroid). Simple radiography and thyroid scintigraphy can also be used. The techniques for physical examination of the thyroid were already described in volume 1, Section I, Chapter 5. Here we will mention a special maneuver to detect endothoracic goiter and measurements to check for thyroid increases.

Marañón maneuver

When an endothoracic goiter is suspected, due to upper thoracic venous circulation, dyspnea, etc., this maneuver is useful, in which, by raising the arms and throwing the head back, the dyspnea and congestion of the face are exaggerated, because the upper orifice of the thorax is narrowed. Its value is fundamentally due to the fact that these signs are lacking in other mediastinal tumors.

Measurements

When an increase in the volume of the thyroid gland is observed, it is advisable to perform measurements at the base of the neck. It is possible to perform them with a compass, to determine the anteroposterior diameter, or with a tape measure, to measure the circumference of said base, as can be seen in figures 71.8 and 71.9, respectively.

mind.

Semiography and semidiagnosis

Let us point out here the characteristics of some types of goiters.

Simple colloid goiter

The thyroid presents a moderate and symmetrical increase in volume. It rarely weighs more than 100 g and shows some increase in consistency (Fig. 71.10).

Nodular goiter or adenomatous goiter

They can be single or multiple and in them the largest sizes of the gland are observed; some cases exceed 1,000 g. They can grow down and behind the sternum, producing a mediastinal syndrome; This form is the so-called endothoracic goiter. On palpation it appears asymmetrical, polylobulated and with increased consistency (see Fig. 71.10).

Iatrogenic goiter

It is produced by the administration of iodides, cyanates, thioureas and other drugs. It behaves like a primary diffuse hyperplasia, although it can sometimes take on a nodular form.

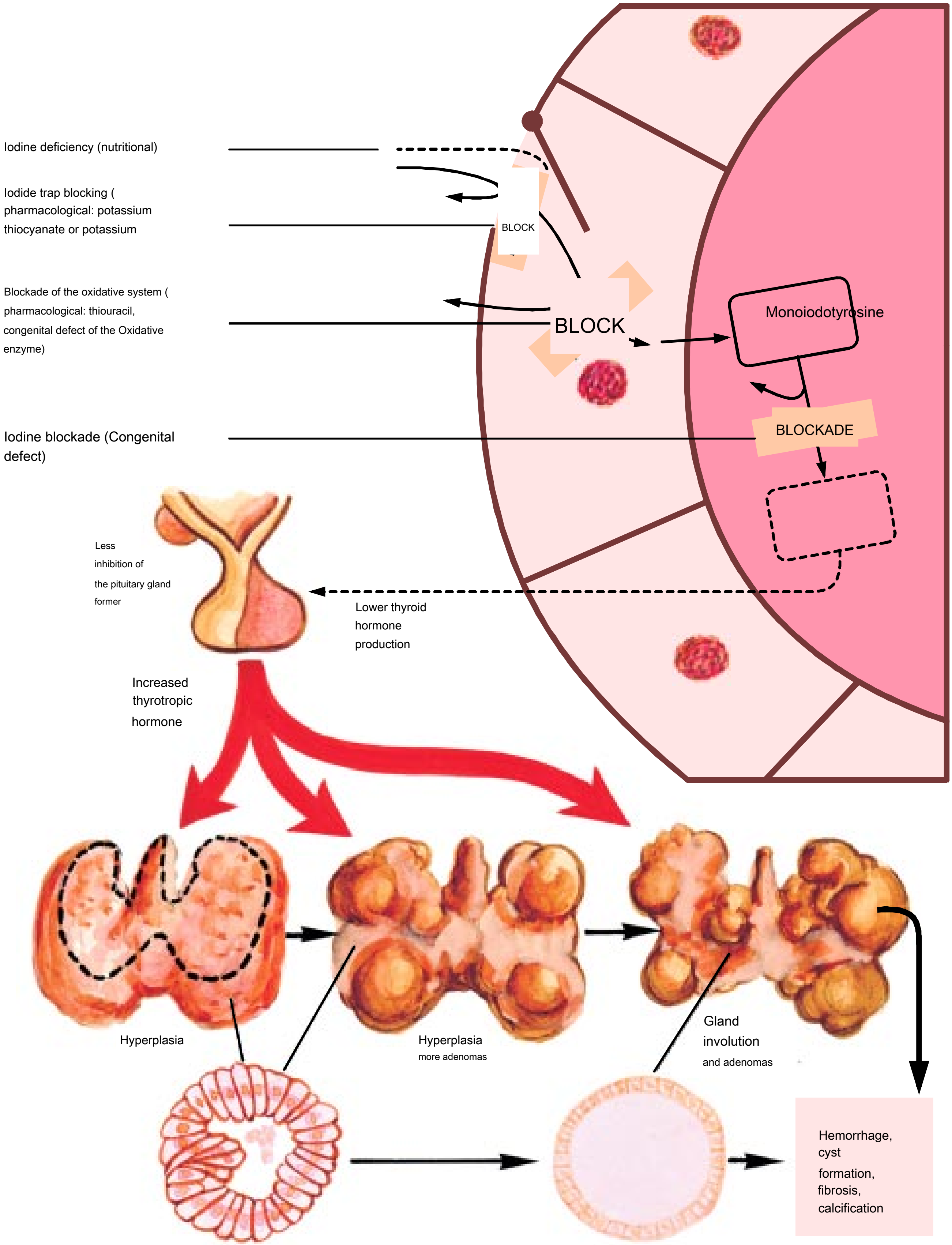

Iodine deficiency (nutritional)
Iodide trap blocking (pharmacological: potassium thiocyanate or potassium
BLOCK
Blockade of the oxidative system (pharmacological: thiouracil, congenital defect of the Oxidative enzyme)
BLOCK
Monoiodotyrosine
Iodine blockade (Congenital defect)
BLOCKADE
Less inhibition of the pituitary gland former
Lower thyroid hormone production
Increased thyrotropic hormone
Hyperplasia
Hyperplasia
more adenomas
Gland involution
and adenomas
Hemorrhage, cyst formation, fibrosis, calcification

Fig. 71.7 Factors causing goiter.

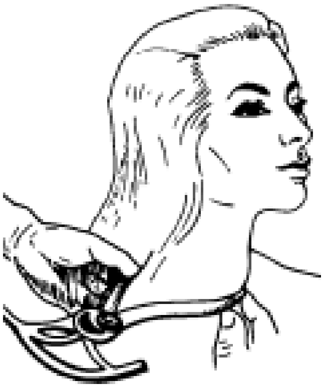


Fig. 71.8 Measurement of the anteroposterior diameter at the base of the neck.

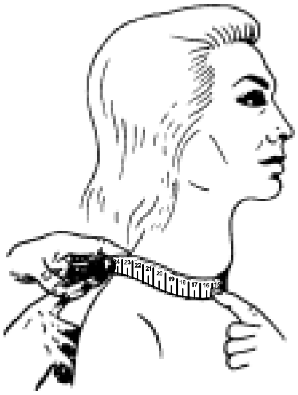


Fig. 71.9 Measurement of the circumference of the base of the neck.

Familial goiter

It is usually multinodular.

Graves-Based disease

It is frequently accompanied by a diffuse, symmetrical goiter, which sometimes reaches 80-100 g.

Acute infectious thyroiditis

The gland is enlarged, it is painful on palpation and also when swallowing; It is accompanied by fever, dysphagia, etc. (fig. 71.11).

Chronic thyroiditis

The two best known ways are:

1. Hashimoto's struma (lymphocytic), which is the most common and is characterized because its gland is symmetrical and its volume can triple. Its consistency is firm like that of rubber.

2. Riedel's struma (fibrous), which is characterized by its variable size, asymmetric gland and stone consistency. Nodules can be seen on palpation.

Figure 71.12 shows the appearance of the gland in both cases.

Neoplasms

They can present themselves as:

1. Adenomas, which are usually solitary and sometimes somewhat
2. Malignant tumors. Among which we have:
   - a) Papillary carcinomas, which range in size from very small, which have been accidentally discovered by surgery, to nodules measuring 10 cm in diameter. They are the most common malignant tumors of the thyroid gland (Fig. 71.13).
   - b) Follicular carcinoma, which varies from an apparently encapsulated nodule, reminiscent of a adenonma, a manifestly cancerous mass that occupies an entire thyroid lobe.
   - c) Medullary carcinoma, with a stone consistency, is sometimes small, although there are some voluminous ones and they can be bilateral and easily palpable.
   - d) Anaplastic carcinoma, which is one of the most malignant forms and whose growth is undifferentiated; It occurs between the seventh and eighth decades of life. Tumor growth is sometimes observed within a week of evolution.

EXOPHTHALMIA OR EXOPHTHALMOS

Concept

It is the abnormal projection or protrusion of the eyeballs. In some cases it is so pronounced that they are not completely covered by the eyelids. Due to ocular protrusion, the upper eyelid is abnormally elevated and the eyes appear too open due to the contracture of the levator of the upper eyelid, which is why the patient's face takes on an expression of alarm or surprise.

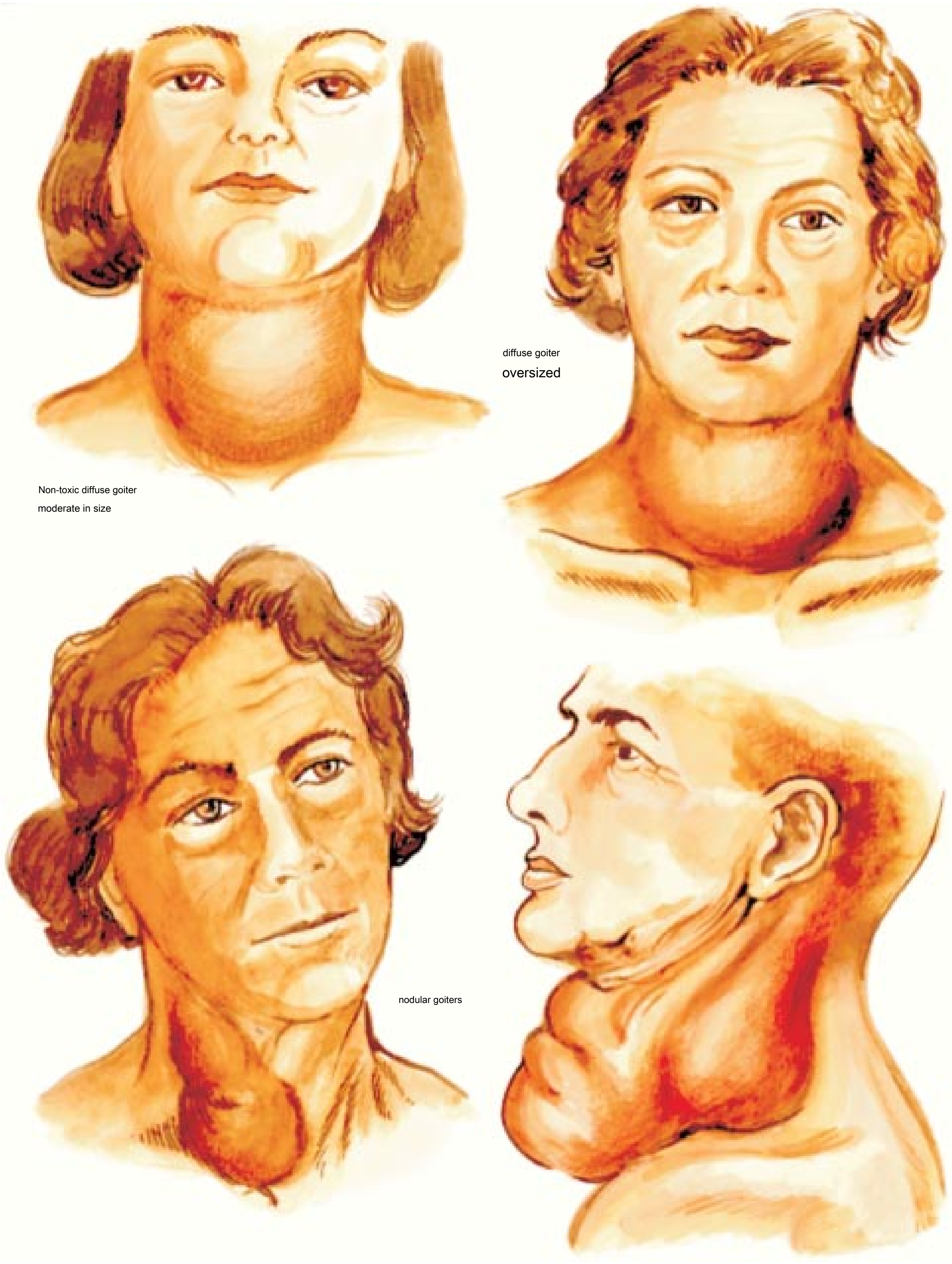
Exophthalmia can be bilateral or unilateral.

Semiogenesis, semiography and semidiagnosis

Bilateral exophthalmia

Its most important cause is hyperthyroidism (Graves-Basedow disease) (fig.71.14). In this disease, exophthalmia is accompanied by other symptoms; goiter, tachycardia, tremor, nervousness, muscle atrophies, as well as a series of ocular signs that make the diagnosis possible (see Chapter 73).

Sometimes the disease is not of the same degree in both eyes and in the initial periods it may be unilateral for a time.

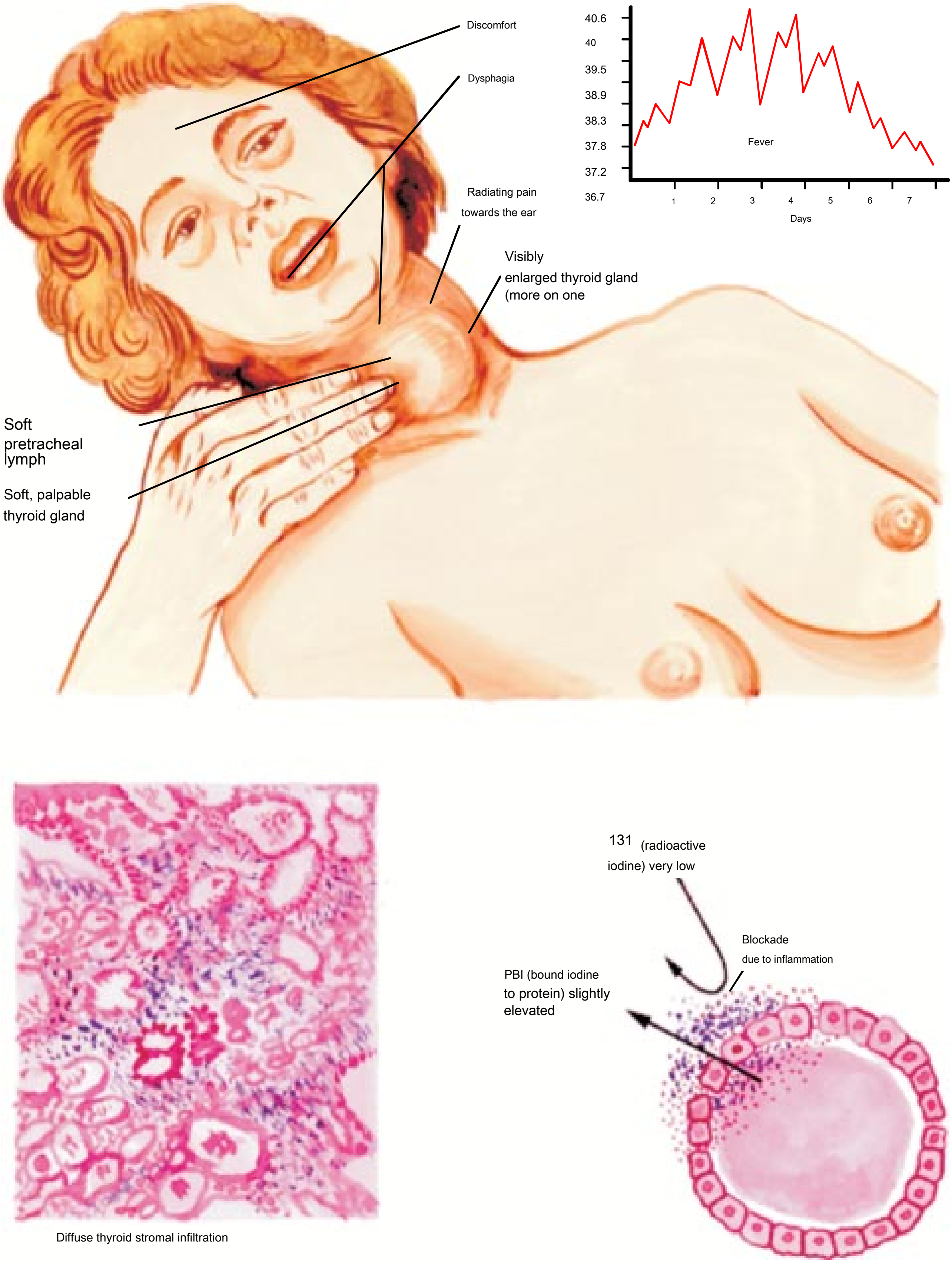


Non-toxic diffuse goiter
moderate in size

diffuse goiter
oversized

nodular goiters

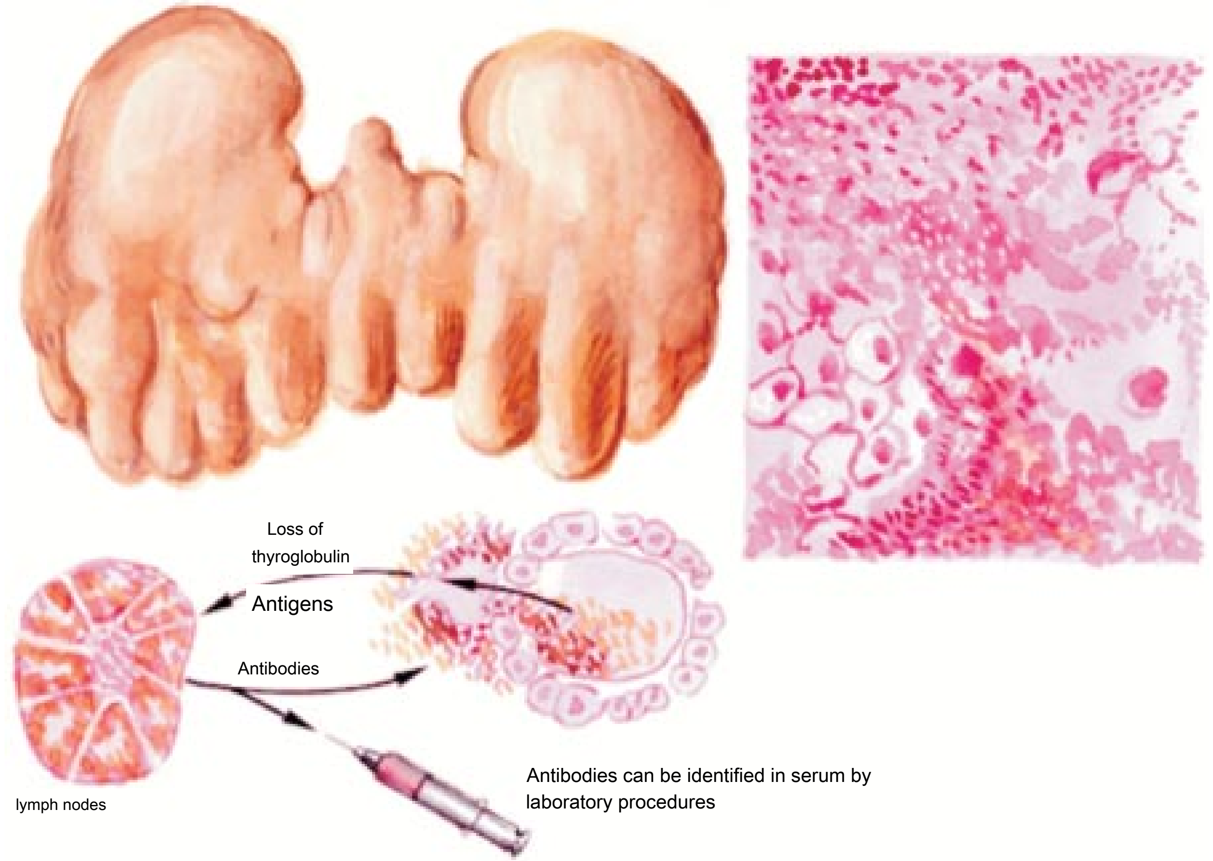
Fig. 71.10 Simple colloid goiter and nodular or adenomatous goiter.


Discomfort
Dysphagia
Radiating pain
towards the ear
Visibly
enlarged thyroid gland
(more on one
Soft
pretracheal
lymph
Soft, palpable
thyroid gland
40.6
40
39.5
38.9
38.3
37.8
37.2
36.7
Fever
1
2
3
4
5
6
7
Days
131 (radioactive
iodine) very low
Blockade
due to inflammation
PBI (bound iodine
to protein) slightly
elevated

Diffuse thyroid stromal infiltration

Fig. 71.11 Acute thyroiditis.

HASHIMOTO STRUMA


Loss of
thyroglobulin
Antigens
Antibodies
lymph nodes
Antibodies can be identified in serum by
laboratory procedures

RIEDEL STRUMA

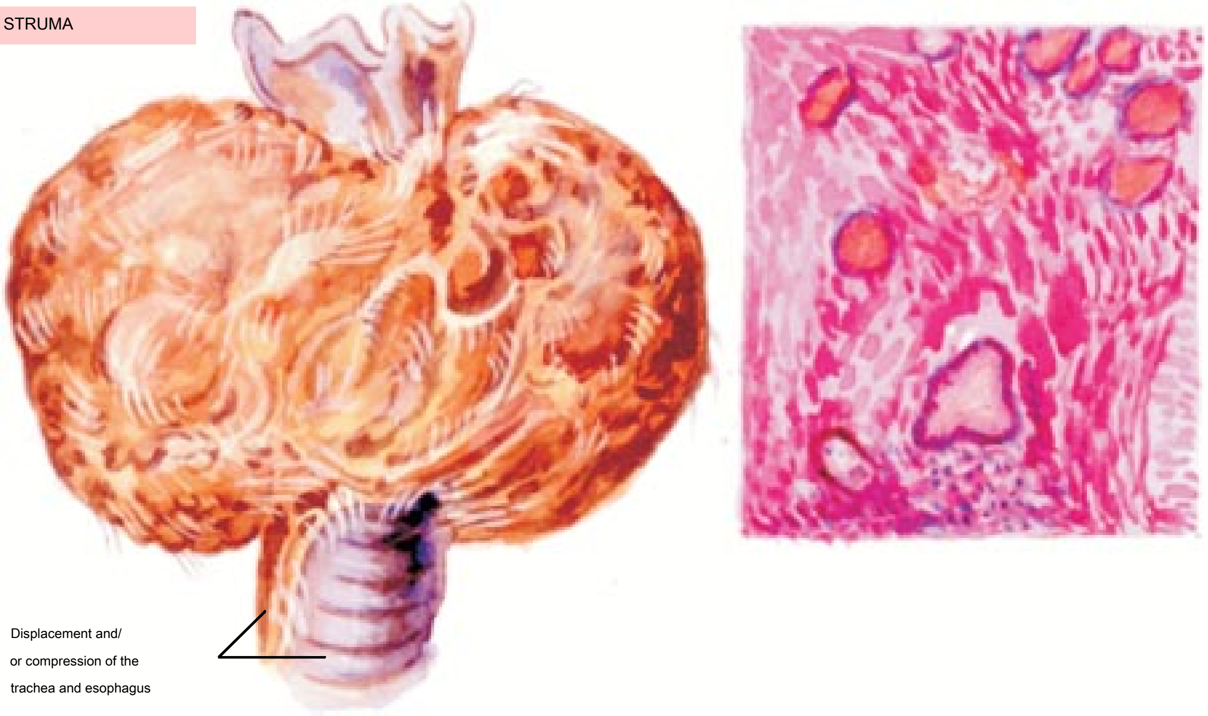

Displacement and/
or compression of the
trachea and esophagus

Fig. 71.12 Chronic thyroiditis.

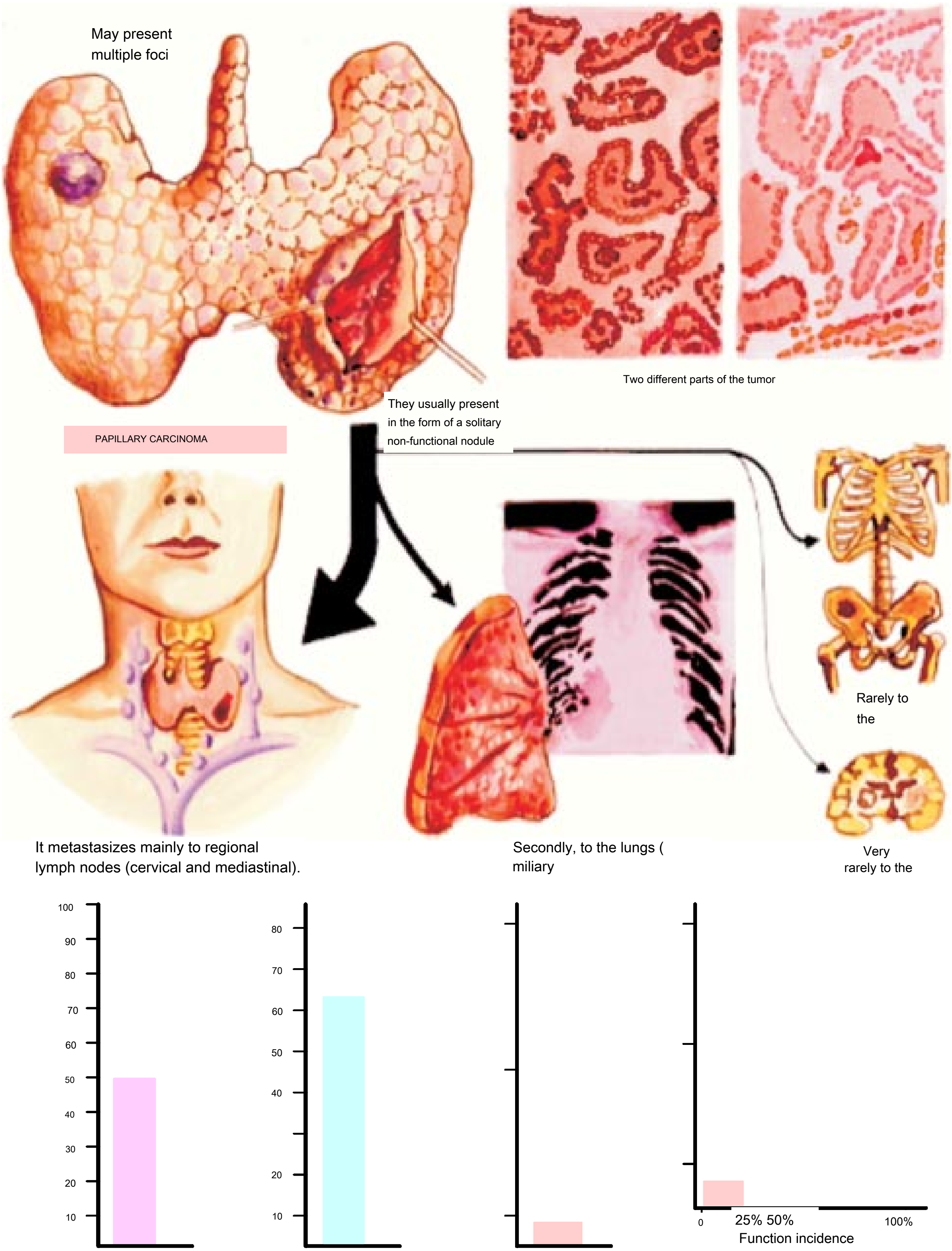

May present
multiple foci
Two different parts of the tumor
They usually present
in the form of a solitary
non-functional nodule
PAPILLARY CARCINOMA
Rarely to
the
It metastasizes mainly to regional
lymph nodes (cervical and mediastinal).
Secondly, to the lungs (
miliary
Very
rarely to the
100
90
80
70
60
50
40
30
20
10
80
70
60
50
40
20
10
0
25% 50%
100%
Function incidence

Fig. 71.13 Papillary carcinoma of the thyroid.

Exophthalmia or exophthalmos can be called malignant, due to the intense protrusion of the eyeballs that does not allow the eyelids to close and exposes the conjunctiva and cornea, which produces erosion of the same and progressive damage that can lead to the loss of vision in that eye. This type of exophthalmia is caused by an autoimmune reaction, in which the antigen may be a segment of thyroglobulin "trapped" in the oculomotor muscles and retro-orbital fat. It has also been related to an exophthalmos-producing substance (EPS), which triggers the immunological process in the orbital tissues.

Sometimes, radioisotopic medical or surgical treatment of hyperthyroid patients aggravates exophthalmia, since, as thyroid secretion stops, TSH increases;

However, the symptoms of thyroid hyperfunction decrease or disappear.

It can also be seen in diabetes insipidus.

Bilateral exophthalmia may also appear in cavernous sinus thrombosis. It generally begins on one side and later spreads to the other, in advanced periods; The eyes project forward and remain fixed, the eyelids are red and swollen, the frontal and ophthalmic veins become prominent and appear dilated.

Unilateral exophthalmia

Generally caused by the following processes:

1. Infectious. Orbital cellulitis, orbital periostitis, tuberculosis, etc.
2. Vascular. Cavernous sinus thrombosis and orbital vein thrombosis.
3. Meningocele, encephalocele.
4. Arteriovenous aneurysm. Accompanied by a pulsatile exophthalmia, which is very evident and in which an intense murmur is heard. Compression of the carotid artery on the same side reduces the heartbeat and intensity of the murmur. The cause is the rupture of the carotid artery inside the cavernous sinus, due to trauma with a fracture of the base of the skull.

SIZE ALTERATIONS

In endocrinology services, cases of children whose height is different from what we consider normal are frequent reasons for consultation.

We include this difference in two large groups: one, whose obvious characteristic is smaller size

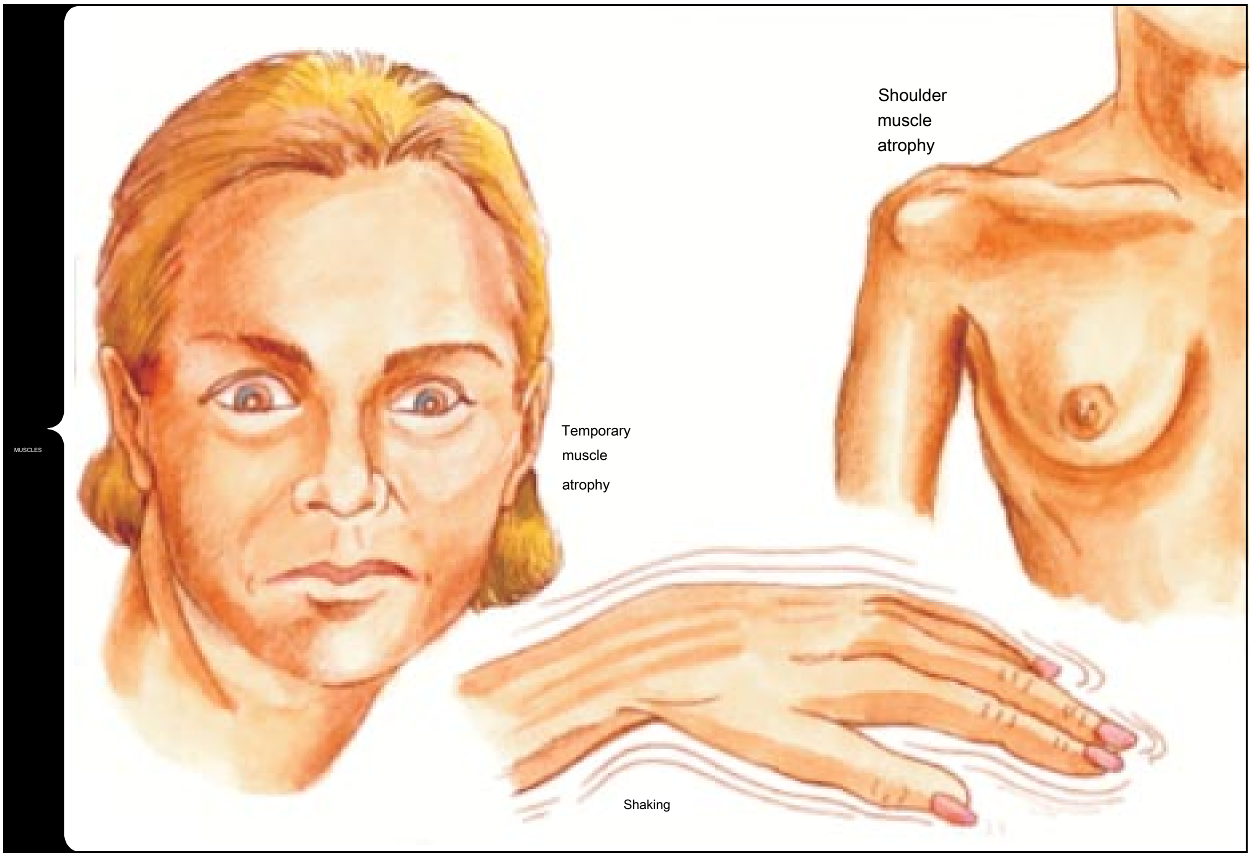


Fig. 71.14 Exoftalmía bilateral, atrofias musculares y temblor en la enfermedad de Graves-Basedow.

(short stature syndrome) and another, which is characterized by having a larger height (tall stature syndrome).

short stature syndrome

Concept

Under this name a series of processes are included whose common link is a smaller size, that is, below the physiological minimum: 1.5 nm in men and 1.38 m in women.

In this syndrome it is necessary to distinguish between dwarfism and infantilism. The limit between dwarfism and short stature is sometimes very difficult to define. Its meaning is very diverse, depending on whether the disorder is accompanied by alterations in the shape or proportion of the different parts of the body or, on the contrary, these proportions are maintained, although on a "small scale." In primary dwarfism the only thing delayed is height, but sexuality (gonads) and psyche are normal. In infantilism, even after puberty, the genital, psychological and height traits typical of childhood persist.

To assess any delay in growth, it is necessary to compare the following with the patient's chronological age:

1. Bone age (skeletal development).
2. Genital age (secondary sexual characteristics).
3. Psychic maturity.
4. Size and weight.

Syndromogenesis or physiopathology

It is established that growth is the progress of the individual in longitudinal dimensions, and represents cell multiplication and the appearance of intercellular substances; Development refers to the increase in the maturity of structures and functions, associated with growth.

In reality, a precise distinction between the two terms is not possible; Their simultaneous use provides a broader idea than the use of any of them separately. Both processes develop simultaneously, continuously, from conception to maturity.

Factors that occur within the organism itself (intrinsic) and factors outside of it or extrinsic intervene in the arrest of growth and development.

Among the intrinsic factors, genetic ones stand out. An anomaly of one or several genes in the parents can lead to different growth patterns in the children. These are fundamentally associated with hormonal factors, produced by the internal secretion glands, with the relevant participation of the nervous system.

Hormones involved in growth and somatic development

1. Pituitary growth hormone (GH).
2. Thyroid hormones: thyroxine, triiodothyronine.
3. Androgens and estrogens.
4. Insulin.

All these hormones have stimulating action.

5. Glucocorticoids.

This group has inhibitory action.

Etiological classification

A. Bone.

1. Achondroplasia.
2. Osteogenesis imperfecta.
3. Chondrodystrophy.
4. Pseudohypoparathyroidism.
5. Rickets.
6. Spine diseases (Pott's disease, osteomyelitis, etc.).

B. Nutritional and metabolic.

1. Deficit of exogenous contributions.
2. Absorption deficit.

C. Deficit in utilization.

1. Deficiency syndromes: nutritional, malabsorption syndrome, celiac disease, cystic fibrosis of the pancreas, etc.
2. Liver disease (childhood cirrhosis).
3. Chronic kidney disease, renal rickets, among others.
4. Chronic infections and parasitism.
5. Hypoxia: congenital heart disease, pulmonary fibrosis, asthma, bronchiectasis, etc.
6. Glycogenosis and mucopolysaccharidosis.

D. Endocrine.

1. Hypothyroidism (the most common): cretinism, infantile myxedema.
2. Hypopituitarisms.
3. Cushing syndrome.
4. Adrenogenital syndrome.
5. Early sexual development.
6. Diabetes insipidus (xanthomatosis).
7. Diabetes mellitus.

E. Genetic.

1.Low family size.
2.Primary:
   a) Without malformations.
   b) With malformations.

3. Chromosomopathies:
    a) Autosomal: trisomy in 21, trisomy in 13 and 15, trisomy in 16 and 18, etc.
    b) Gonosomal: gonadal dysgenesis (Turner syndrome)

E. Constitutional delay in growth and development.

G. Otros.

1. Neurological conditions.
2. Emotional disorders.

Therefore, before affirming the endocrine nature of short stature, it is necessary to rule out the other causes mentioned above, congenital or acquired. It is also necessary to take into account the size of the parents and the race (pygmies).

We will describe some of the clinical conditions already mentioned.

Pituitary short stature syndrome

It is due to a failure in the production of growth hormone. In childhood it determines poor development sexual (arrest in childhood stage), proportional but small shapes and normal intellect. They have significant delay in skeletal development. If the age of these subjects were not known, they would be considered normal.

If the cause is a craniopharyngioma, neurological symptoms arise: headache, hemiparesis, epilepsy, papilledema, etc. X-ray of the sella turcica can establish the diagnosis by discovering the calcified suprasellar tumor.

True dwarfism0 nanosomia

It is due to a lesion with hypofunction of the anterior lobe of the pituitary gland, mainly of the eosinophilic cells that produce growth hormone. Other authors propose a disorder of receptivity of the growth cartilages towards said hormone.

The lack of somatic development affects the different parts of the body equally, that is, there is very short stature with normal proportions.

The subjects are born small and develop normally at the corresponding time: ossification of the growth cartilages, sexual maturation and appearance of secondary characteristics, libido, etc.

In short, only the height remains reduced, which is lower than that corresponding to individuals of the same age and sex. This is a miniature of a normal subject. In the anamnesis there is homologous inheritance.

Hypothyroid dwarfism: congenital myxedema and sporadic cretinism (Fig. 71.15)

It is observed when the loss of thyroid function occurs in full somatic growth. En la hipoplasia or congenital aplasia of the thyroid, the so-called infantile myxedema can be seen. When thyroid deficiency occurs at the beginning of puberty, in a previously healthy child, we speak of infantile or juvenile hypothyroidism, also accompanied by short stature.

The first manifestations of congenital thyroidism are observed a few months after the child is born or when the child passes into early childhood. The condition is translated by: delayed skeletal development, especially of the long bones; the fontanels remain abnormally open; teeth small, uneven and late; and there is a delay in sexual and psychological development, since the child does not speak and is indifferent to psychic and emotional stimuli. This is what is called myxedema idiocy. There is also motor delay (hypotonia, difficult ambulation), altered dermal trophicity (dry, infiltrated, scaly skin, lacking sweat, and cold) and the hair is sparse, coarse, and light in color.

When the disorder occurs after puberty, the symptoms are less noticeable and begin with an arrest in physical and mental development. The skin then becomes pasty and pale, with myxedema infiltration on the backs of the hands and wrists, as well as on the ankles and lower eyelids; hair and nails grow slowly; the voice is hoarse; there is a tendency towards obesity; The character is placid and indolent.

Endemic cretinism

It is a special variety of hypothyroid dwarfism; It occurs collectively in some mountainous regions, particularly the Swiss Alps, the Himalayas, the Pyrenees, the Andes, etc. Etiologically, it is undoubtedly related to the lack of iodine in the water drunk in those regions.

Iodine deficiency prevents the normal synthesis of thyroxine and this allows hypersecretion of the thyrotropic pituitary hormone, which is goitrogenic. In fact, the condition is usually accompanied by a goiter alteration of the thyroid gland whose genesis is prenatal.

Some cretins reach old age; the majority die young.

Achondroplasia or chondrodystrophy

It's another kind of dwarfism. In chondrodystrophy, already in intrauterine life there is an anomaly of the connecting cartilages that disrupts the longitudinal growth of the extremities. The head and trunk, however, remain normal (see Fig. 24.10, in volume 1, Section II, Chapter 24). Chondrodystrophy manifests itself in the newborn, in such a way that those who suffer from it maintain a great resemblance to each other, due to their grotesque appearance: the disproportionately large head, with sunken nasal root; excessively short limbs

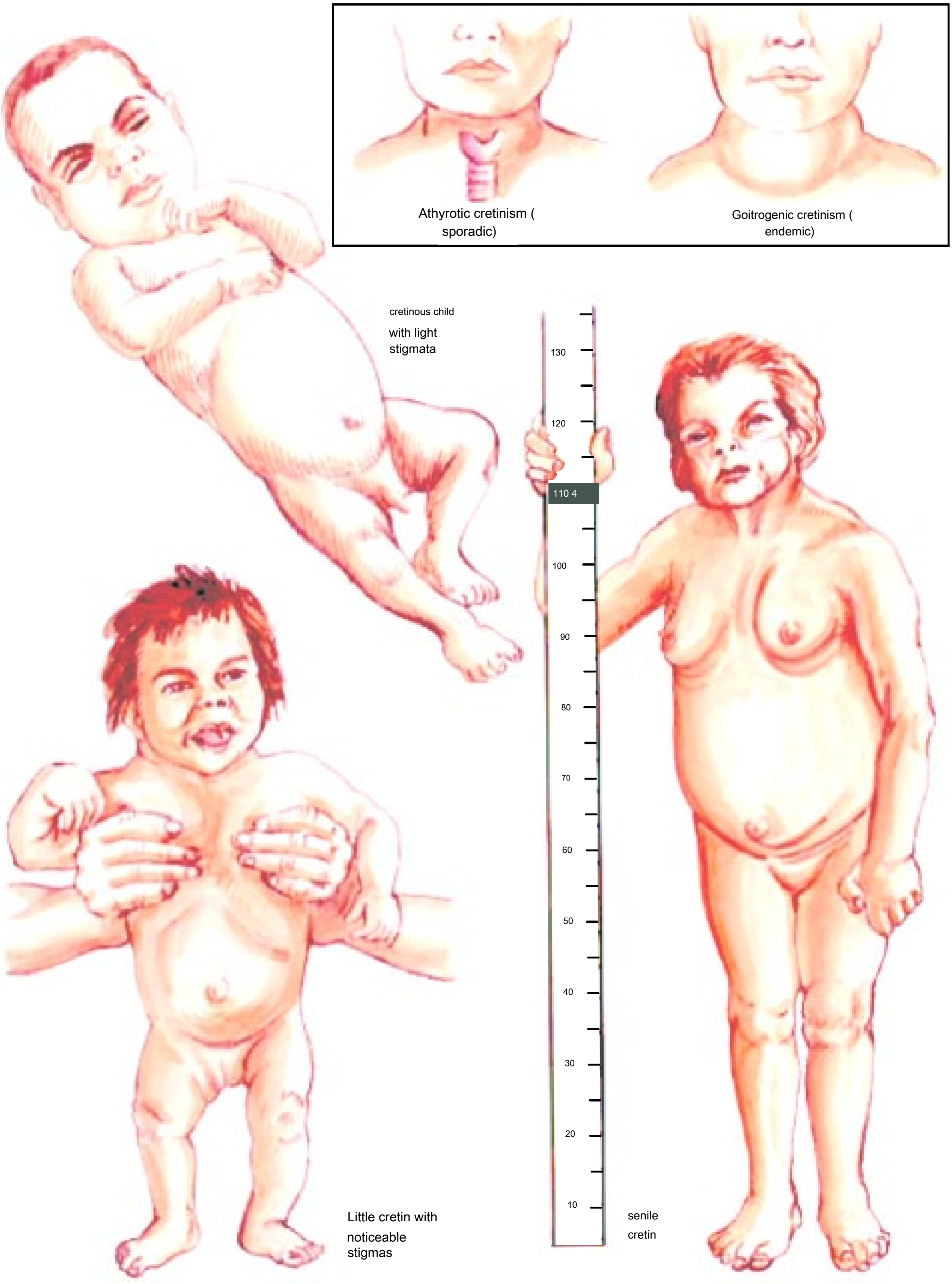

Athyrotic cretinism (sporadic)
Goitrogenic cretinism (endemic)
cretinous child with light stigmata
130
120
110 4
100
90
80
70
60
50
40
30
20
10
Little cretin with noticeable stigmas
senile cretin

Fig. 71.15 Hypothyroid dwarfism: congenital myxedema and cretinism.

(micromelia); wrinkled skin, with wide folds; the muscles develop vigorously; hands short and wide, square in shape, with the three central fingers of equal length (trident fingers); genital organs well developed, sometimes excessively; intelligence clear, even well developed.

This type of dwarfism is hereditary, sometimes dominant and other times recessive.

Low pituitary size with hypogonadism

It is a syndrome characterized by the inhibition of the development of the entire organism at any stage of childhood, that is, from the time the child walks until puberty. Presents: small size, with little genital development (infantile penis, small testicles, sometimes unilateral or bilateral cryptorchidism); absence of secondary sexual characteristics (lack of hair on the pubis and armpits); persistence of infantile lanugo on the back and extremities; and, sometimes, congenital malformations (hypoplasia of the last phalanges with curvature of the ends of the little fingers).

There are clinical forms with associated deficiency of gonadotropic, thyrotropic and corticotropic hormones, which present the complete picture of hypopituitarism.

Short stature with Turnerian gonadal dysgenesis (gonadal dysgenesis or Turner syndrome)

There is ovarian aplasia with pituitary integrity.

It is characterized by poor development, hypoplastic external genitalia, absolute primary amenorrhea and lack of breast development; In addition, various malformations: short and wide neck, pterygium colli (skin folds that extend from the base of the skull to the shoulders) and bilateral cubitus valgus.

This is a form of female hypogonadism, which will be discussed in greater detail later (Chapter 77).

tall stature syndrome

In reality, this syndrome is rare. It constitutes less family concern, except in children with very tall stature or who have another alteration.

We have to consider two aspects in this syndrome: one static and the other dynamic; In this, height and growth speed are respectively greater than chronological age according to race and sex.

Etiological classification

A. Nutritional. Associated with exogenous obesity.

B. Endocrine.

1. Pituitary (gigantism, acromegaly).
2. Precocious sexual development and adrenogenital syndrome.
3. Hyperthyroidism.
4. Hypogonadal.

C. Hypothalamic.

1. Congenital generalized lipodystrophy.

D. Genetic.

1, High family size (constitutional):
  a) YY syndrome.
  b) Klinefelter syndrome.
  c) Macrofetuses (in diabetic mothers).

E. Others.

1. Neurofibromatosis.
2. Marfan syndrome.

There is also local gigantism, which is observed in: acromegaly, arachnodactyly, gigantism of a single limb or a finger, macrodactyly, etc.

Of this classification, we will only describe the endocrine ones.

Pituitary gigantism

It is caused by excess growth hormone in the child. He has a tall height for his age and an excessive growth rate, which includes the soft parts; there are acromegaloid features (see Fig. 24.9, volume 1, Chapter 24).

Precocious sexual development and adrenogenital syndrome

They produce tall stature during childhood due to excess androgens, but frequently the early welding of the epiphyses interrupts growth before normal adult height is reached, which actually produces a syndrome of short pubertal stature.

High stature with hypogonadism

Primary gonadal atrophy, castration due to tumors, secondary atrophy due to diseases that occurred before epiphyseal closure, produce a characteristic type of eunuchoid gigantism, with long limbs.

Congenital generalized lipodystrophy

Possibly diencephalohypophyseal etiology. More common in females. These cases present at birth with rare facies, generalized lipodystrophy (all adipose tissue is missing), marked muscular relief, large hands and feet. There is hepatomegaly, hyperlipidemia, acanthosis nigricans, prominent navel, accelerated growth rate and bone and dental development.

In adults, bone cysts, polycystic ovaries and diabetes mellitus not associated with ketosis occur.

Cerebral gigantism 0 Sotos syndrome

There is mental retardation, acromegaloid-type facies, high-arched palate and marked kyphosis.

HIRSUTISM

Concept

It is the increase in the female's body hair, in areas that are not usual for her sex. Therefore, it is a syndrome that only has clinical expression in women and is characterized by increased body hair and a change in its characteristics, in the places corresponding to men.

Hair categories

Hair or hair is divided into three categories:

1. General body hairs, which cover the limbs and trunk and begin their development from childhood.
2. Ambisexual hair: axillary and pubic hair. They appear at puberty and are common to both sexes.
3. Male sexual hairs: beard, mustache, pretragus region, anterior chest hair, suprapubic hair, which transforms the ambisexual pubic triangle into a rhombus whose elongated vertex follows the white line. The woman does not have, in a normal state, male sexual hairs.

Hirsutism must be distinguished from simple hypertrichosis, which is an excess of body hair, but respecting the normal topography of women.

Semiogenesis or pathophysiology

Hair production is closely linked, on the one hand, to the secretion of androgenic hormones, on the other, to an increase in the hormonal sensitivity of the hair follicle to androgens and, finally, to neurovegetative regulation.

Androgen ovens include:

1. Testosterone metabolites (especially androsterone), whose main source is the testis, but it is known that the ovary is also capable of secreting testosterone. This secretion can, in a pathological state, become considerable.

2. The metabolites of the androgen group of adrenal cortico-adrenal hormones constitute two-thirds of the urinary androgens in men and almost all of the physiological androgens in women.

There seems to exist, in a normal state, a certain threshold of these metabolites, from which each category of hair appears; The highest threshold belongs to male hairiness.

A neurovegetative regulation suppresses, however, all the rigor of this parallelism. Nervous factors, of local or central origin, can in fact modify the sensitivity of the pillar receptor and cause, for it, hormone content, a very different abundance of hairiness.

Therefore, hirsutism may be due to hypersecretion of adrenal and ovarian androgens, or it may be mixed.

This hypersecretion can be isolated or accompanied by other steroids, as in Cushing's syndrome (excess cortisol predominates, produced by "carryover" due to the elevation of other steroids).

Examples of these mechanisms are adrenogenital syndromes caused by congenital adrenal enzyme defects or polycystic ovary syndrome.

In summary, in the pathophysiology of hirsutism it is necessary to distinguish:

1. A hormonal sector that includes the adrenal corticosteroids, the gonads and the pituitary gland (which ensure the production of androgens).
2. A nervous sector that includes the hypothalamic (or better, corticohypothalanmic) centers and the neurovegetative system. This sector regulates, on the one hand, the activity and harmony of the hormonal sector and, on the other hand, the sensitivity of the pillar receptor.

Hirsutism appears, in this conception, as a hypertonic manifestation of the pillar function, whose etiology may depend on a primitive lesion of one of the links of the hormonal sector or a disturbance of the nervous direction.

semiography

Clinical picture

Hirsutism is not accompanied by androgenization and occurs in isolation.

When it is accompanied by androgenization, it is a function of age. In young people after puberty and in women.

Here the excess of androgens is manifested by three groups of symptoms that translate: one, the defeminization of the organism; others, masculinization; and others, general somatic and psychic modifications.

Defeminization of the organism

Disturbances of the menstrual cycle constitute its earliest testimony: oligomenorrhea (decrease in menstrual flow), which sometimes gives rise to periods of amenorrhea lasting several months and even true secondary amenorrhea.

Another frequent testimony is primary or secondary infertility, linked to an inhibition of ovulation or the interruption of pregnancies between the second and fifth.

to month.

Finally, intense virilisms can be revealed by breast regression.

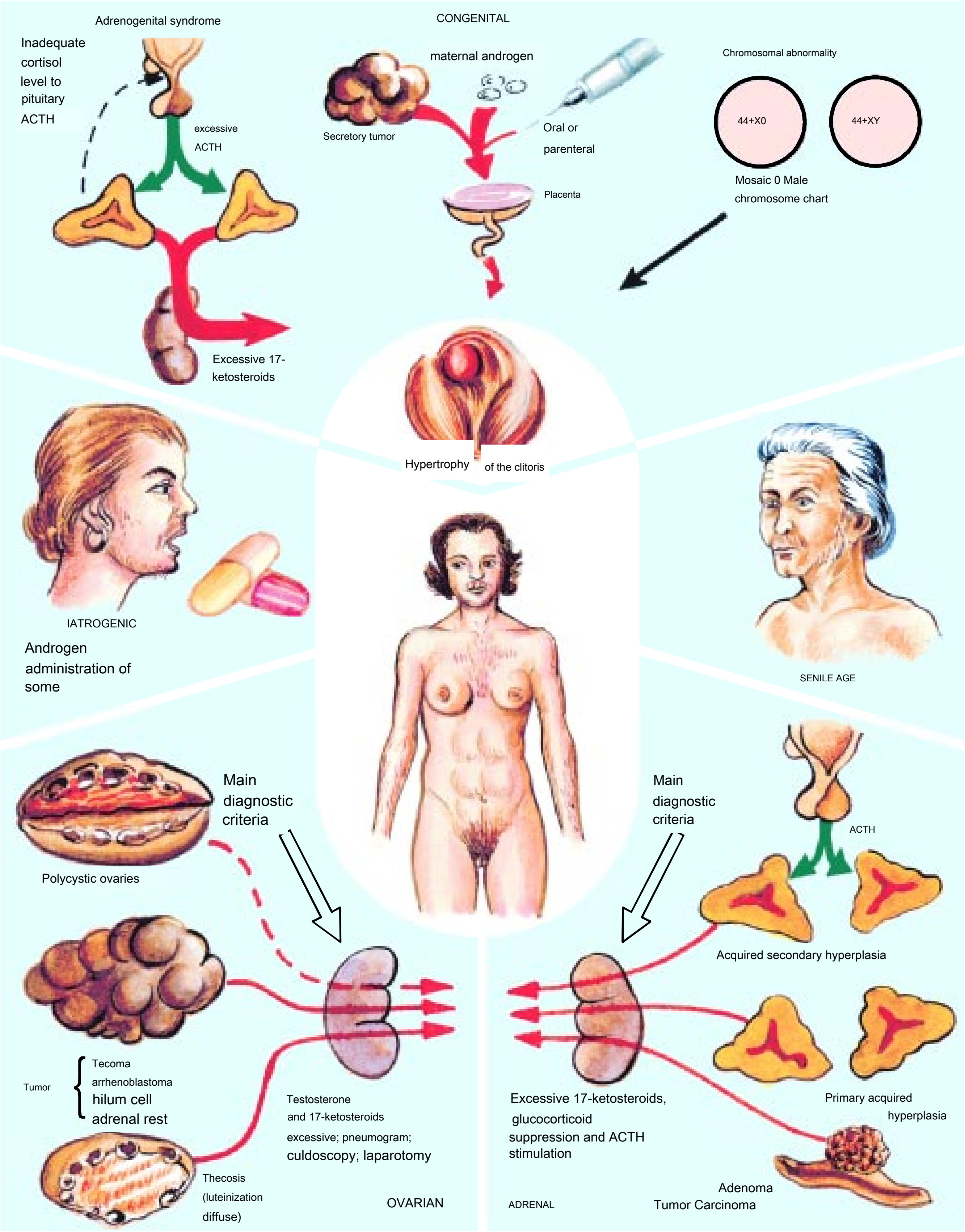

Adrenogenital syndrome
Inadequate cortisol level to pituitary ACTH
excessive ACTH
Excessive 17-ketosteroids
CONGENITAL
maternal androgen
Secretory tumor
Oral or parenteral
Placenta
Chromosomal abnormality
44+X0
44+XY
Mosaic 0 Male chromosome chart
Hypertrophy of the clitoris
IATROGENIC
Androgen administration of some
SENILE AGE
Polycystic ovaries
Main diagnostic criteria
Main diagnostic criteria
ACTH
Acquired secondary hyperplasia
Tumor
Tecoma
arrhenoblastoma
hilum cell
adrenal rest
Thecosis (luteinization diffuse)
Testosterone and 17-ketosteroids excessive; pneumogram; culdoscopy; laparotomy
OVARIAN
Excessive 17-ketosteroids, glucocorticoid suppression and ACTH stimulation
ADRENAL
Primary acquired hyperplasia
Adenoma
Tumor Carcinoma

Fig. 71.16 Hirsutism.

Masculinization of sexual receptors

Hirsutism is its most common expression. The hairs take over normally hairless territories: the face (sideburns, chin, upper lip), the intermammary region and areola of the breasts, the umbilicopubic line, the perianal region and the upper inner part of the thighs.

Associated symptoms

Entries appear on the scalp in the temporal regions. The skin changes, becomes thicker and more oily (seborrheic); The face and upper back may become covered in acne.

At the level of the external genital organs, the labia majora become more voluminous and pigmented, and above all, the clitoris hypertrophies and can achieve a pseudopenile appearance.

Changes in the voice attract attention more quickly, especially in certain professions (singers, telephone operators): the voice drops by one or two pitches and becomes hoarse.

Finally, muscle masses accentuate their prominence.

Morphotype and psyche modifications

The silhouette is modified: the hips lose their rounded contour and the gluteal muscles flatten; The shoulder girdle widens and the biacromial diameter can exceed the bitrochanteric diameter.

The psyche is not spared either: feminine sensitivity is dulled, as is maternal feeling: the character becomes more authoritarian, concerns change, orienting themselves towards sports and business. Sometimes true psychoses, depressive episodes and confusional states with melancholy arise.

Semiodiagnosis (fig. 71.16)

1. Hirsutisms of hormonal origin. They are the rarest but also the most characteristic. It may be an injury:

   a) Of the adrenal cortex: benign or malignant tumor; simple hyperplasia, usually congenital. These injuries can determine:

   Cushing's syndrome: global hypercorticism associated with virilism, signs of hypersecretion of the other groups of adrenal corticohormones (faciotroncular obesity, purple streaking, arterial hypertension and painful osteoporosis).

   Adrenogenital syndrome, Apert-Gallais type: a dissociated hypercorticism, which more particularly affects androgen production and is characterized by more or less isolated virilism.

   b) From the ovary: malignant virilizing tumor, arren0-blastoma, luteoma, hilar cell tumor. They give a picture of pure virilism, associated with amenorrhea: Stein-Leventhal syndrome (see Chapter 77).

   In the two previous cases, a) and b) with pilar virilism, the association of obesity and menstrual disorders is observed: amenorrhea that alternates with menstrual hemorrhages, sterility and, above all, very severe menstrual pains.

   c) Of the pituitary gland: Cushing's disease.

2. Hirsutism of central origin. They are much more common, but their clinical picture is less precise. The disturbance of the central direction is sometimes of organic origin due to:

   a) Meningoencephalitis of the base. It seems that this possibility is far from being exceptional, and is associated with obesity and primary amenorrhea.

   b) Internal frontal hyperostosis. It constitutes the Morgagni-Morel syndrome, characterized by the association of virilism, obesity and psychological disorders.

   COs.

3. Hirsutisıno of medicinal origin. Due to administration of drugs, such as cobalt, diazoxide, diphenylhydantoin, androgens, progesterone, anabolics, corticosteroids, chorionic gonadotropin, etc.

ENDOCRINOMETABOLIC SYSTEM. PITUITARY AND PITUITARY SYNDROMES

72

ANATOMY AND HISTOLOGY OF THE PITUITARY

The pituitary gland or pituitary gland was first described in 1543 by Vesalius. It constitutes the highest endocrine gland in both the functional and pathophysiological order. It directs the wonderful endocrine concert and is closely linked and united to the diencephalon.

The pituitary gland, hypothalamus and vegetative centers form the so-called neurohormonal system.

It is ovoid in shape and weighs between 0.6-0.8 g in the adult; The average length of their diameters is 6-8 mm in the anteroposterior diameter, 6-8 mm in the vertical diameter, and about 12-15 mm in the transverse diameter.

Apparently there is a certain relationship between the size of the pituitary gland and the height of the individual.

It is significant that this gland is slightly larger in the woman who has given birth than in the man or the pubescent woman.

Age also seems to be a factor in variations in weight and size of the pituitary gland, since an increase occurs during middle age after which an overall decrease occurs.

The glandular portion of the human pituitary gland comes from an ectodermal outpouching of the oropharynx called Rathke's pouch.

The cephalad extension of this pouch puts it in contact with the primitive diencephalon of the embryo. This contact zone of the central nervous system is destined to become the posterior lobe or neurohypophysis by differentiation.

Less clearly defined in man, the portion of Rathke's pouch in direct contact with the neurohypophysis forms the pars intermedia (intermediate part).

The part of Rathke's pouch that is not in contact with the diencephalon grows and gives rise to the anterior lobe or adenohypophysis.

In humans, the pars intermedia is not fully developed, the cells are scattered in the anterior lobe.

The cephalic portion of Rathke's pouch is separated from the connection with the oropharynx by a penetrating growth of the mesenchyme, but remnants of the lower portion of the pouch, which form the human pharyngeal pituitary, are often included in the oropharyngeal duct.

The neurohypophysis preserves connections with the base of the brain, which form the pituitary stalk or pedicle. This nervous connection is crucial for hypothalamic regulation of adenohypophyseal function.

The pituitary gland is located in a kind of depression that exists at the level of the sphenoid bone (fig. 72. 1). Its bed is part of the roof of the sphenoid sinus and its four upper extremities rise to constitute the anterior and posterior clinoid processes, giving rise to this configuration for this depression to be known as the sella turcica, due to the resemblance it has to it. The depression can be oval, round or flattened and is covered by the dura mater and its roof is covered by a division of the dura mater called the sella diaphragm. Through this you can see an opening through which the pituitary stalk passes. In this way, the pituitary gland is attached to the bottom of the infundibulum by a type of stem that extends downwards and backwards and is called tuber cinereum.

The cavity of the infundibulum is nothing more than an extension of the cavity of the tuber cinereum and, therefore, is part of the third ventricle.

The measurement of the sella turcica is probably of greater practical importance than the actual dimensions of the gland. In normal adults, the anteroposterior diameter is 17 mm, the vertical diameter is 14 mm, and the width is about 19 mm. The calculated lateral area is 130 mm? and the volume of 1 100 mm². In general, the gland is proportional to the dimensions of the bone receptacle. Tumors of the gland or neighboring regions can cause erosion, enlargement or distortion of the sella turcica that can be recognized by radiographic means, as well as suprasellar calcification, demineralization or depression of the floor of the sella, or spherical and enlarged shape of the fossa.

The cavernous venous sinuses are located on each side of the sella turcica and communicate with each other, both in front and behind, through the intercavernous sinuses, which cross the anterior edge of the diaphragm of the sella turcica.

Next to the pituitary gland and on each side of it are the oculomotor nerves (I pair), pathetic nerve (IV pair) and the ophthalmic branch of the trigeminal nerve (V pair), which run along the external wall of the cavernous sinus, and the abducens nerve (VI pair), which runs inside the cavernous sinus (see Fig. 15.3, volume 1, Section I, Chapter 15).

In front and above the pituitary gland and separated from it by the diaphragm of the sella, is the optic chiasm. These intimate neighborhood relationships are very important in clinical and surgical settings when considering the compressive effects caused by the growth of the gland and this explains why symptoms and visual disturbances are frequently observed in cases of pituitary tumors.

PORTAL CIRCULATION OF THE PYPOPHYSIS

In contrast to the connection or relationship between the hypothalamus and the neurohypophysis, it has not been possible to demonstrate the existence of nerve fibers that, starting from the central nervous system, penetrate the adenohypophysis. However, sectioning the pituitary stalk at different levels has not abolished the gonadotropic function of the pituitary.

We must accept another existing mechanism other than the neurogenic one and in this case we will have to recognize the vascular mechanism based on the hypothalamic-pituitary circulation.

The hypothalamic and pituitary arteries are completely independent and there is no vascular communication between the supraoptic and paraventricular nuclei and the adenohypophysis (see Fig. 72.1l). The adenohypophysis and neurohypophysis are linked by a large number of capillaries (sinusoids) and portal vessels (Fig. 72.2).

The arteries of the pituitary gland are: the superior and inferior pituitary glands. The superior ones arise from the internal carotid and the posterior communicating artery, they branch and are distributed in very small capillary branches in the stem and in the pars distalis of the adenohypophysis, forming sinusoidal plexuses.

The inferior pituitary arteries arise from the internal carotid, penetrate the neurohypophysis and end in capillaries within it.

The sinusoidal plexiform capillaries of the pars tuberalis of the adenohypophysis are seen to penetrate the stalk of the adenohypophysis.

There are also, on the edge of the median eminence, numerous sinusoids that are connected, through very fine vessels, with the capillaries of the hypothalamus and pituitary gland, circulating from top to bottom, that is, from the hypothalamic region to the pituitary gland.

The veins of the pituitary gland are of two types: portal veins and systemic veins. The portals accompany the branches of the superior pituitary artery and come from a capillary plexus found in the adenohypophysis and from the confluence of small and delicate capillaries on the surface of the pituitary stalk.

These capillaries penetrate into the stem at regular intervals and connect the superficial capillaries with the deep ones. From these two systems the course of the plexus of portal vessels can be followed to the adenohypophysis where blood is poured into the sinusoids before passing into the systemic circulation.

After passing through the sinusoids, blood passes into the pituitary veins, which drain into the cavernous sinus.

It is through this circulatory system that the neurohormonal mechanisms that influence the secretion of the adenohypophysis are established.

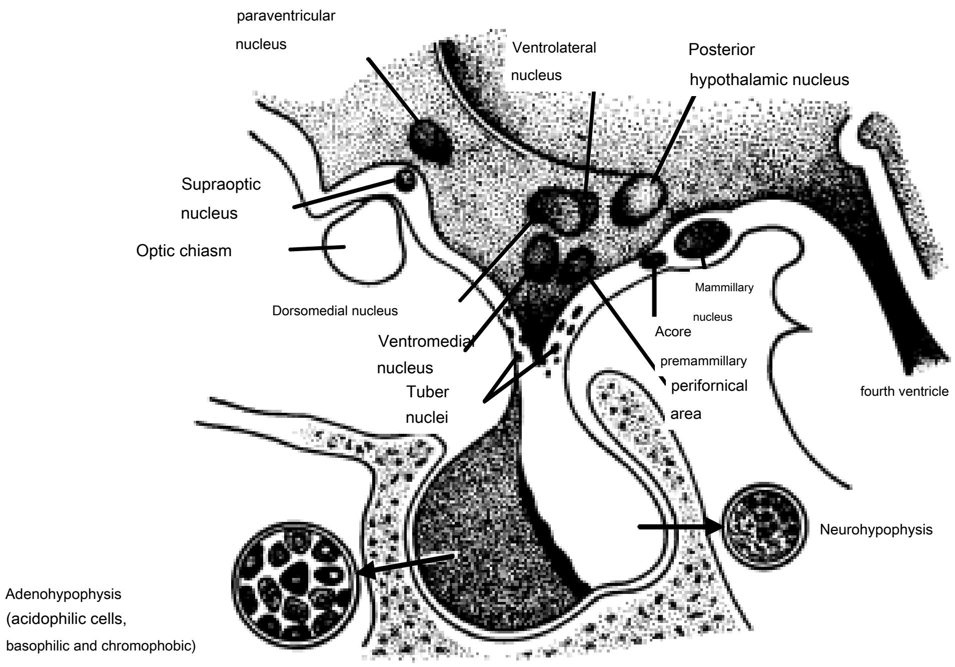

paraventricular nucleus
Ventrolateral nucleus
Posterior hypothalamic nucleus
Supraoptic nucleus
Optic chiasm
Dorsomedial nucleus
Ventromedial nucleus
Tuber nuclei
Mammillary nucleus
Acore premammillary perifornical area
fourth ventricle
Neurohypophysis
Adenohypophysis (acidophilic cells, basophilic and chromophobic)

Fig. 72.1 Anatomy of the pituitary gland.

RESULT OF HYPOPHYSECTOMY

The experimental removal of the pituitary gland demonstrates the extraordinary influence that its hormones exert on the rest of the endocrine glands and on the human body in its development.

With the advancement of surgical techniques, it becomes possible to remove the gland without damaging the brain tissue and the results obtained can be evaluated in their entirety.

dwarfism. After removal of the gland there is an immediate and almost complete arrest of the animal's growth. It is proven that the absence of the anterior lobe causes dwarfism, while the absence of the posterior lobe alone does not produce it. Furthermore, maturation stops in the animal, maintaining its infantile characteristics.

Effects on reproduction. In the female animal there is a marked organic and functional reduction.
In men, there is a notable regression of the reproductive system, with loss of libido.

Decreased metabolism and body heat. Basal metabolism is low, lower than in thyroidectomized patients, and body temperature drops markedly.

Atrophy of the thyroid and adrenal glands. The thyroid is extremely reduced in size and atrophic changes occur in the follicular epithelium.

The adrenal glands also show marked atrophy, especially the cortex being the most affected part. If the posterior lobe is removed in isolation, these changes do not occur, which indicates that the atrophic phenomena, first, and then the functional ones, are caused by the deficient secretion of the anterior lobe.

Other phenomena. Decreased weight of the kidneys, spleen and liver, decreased resistance to cold, infections and external aggressions; hypoglycemia and increased insulin sensitivity. In animals, if they are pregnant, abortion occurs, and the cessation of lactation can be demonstrated.

HISTOLOGY OF THE PITUITARY

Given the fact that the pituitary gland produces a certain number of hormones that depend on the cellular structures, the need to study this cytology is imposed.

According to classical notions, the adenohypophysis consists of three types of cells: acidophilic, basophilic and chromophobic, which are clearly differentiated with special techniques.

Acidophilic (eosinophilic) cells form approximately 40% of the adenohypophysis. They are polygonal cells with a small nucleus and cytoplasmic granules. Under the electron microscope, two types of acidophilic cells can be differentiated: some, completely granulated (corresponding to the growth hormone) and others, which have few but thick granules (which seem to correspond to prolactin).

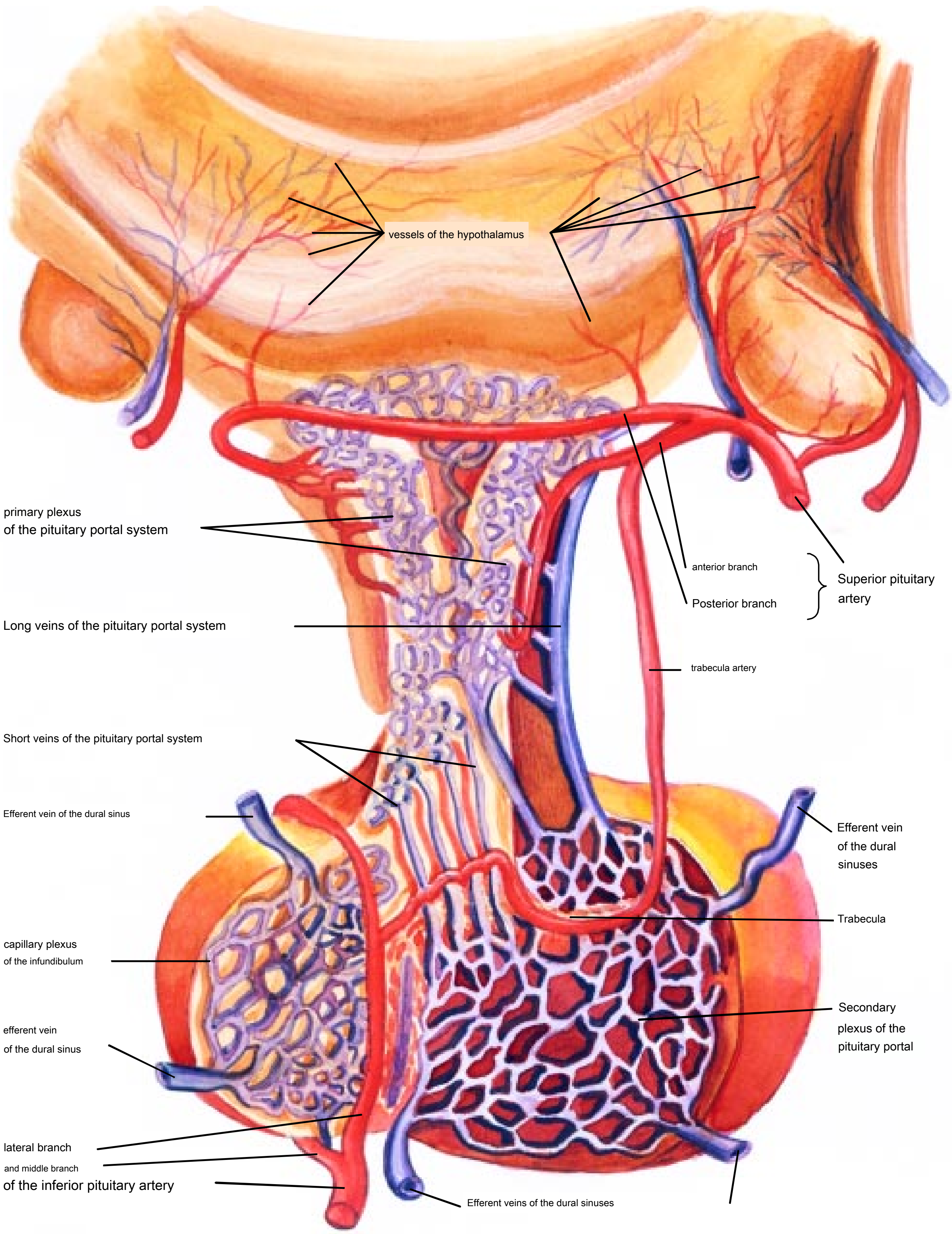

vessels of the hypothalamus
primary plexus
of the pituitary portal system
anterior branch
Posterior branch
Superior pituitary
artery
Long veins of the pituitary portal system
trabecula artery
Short veins of the pituitary portal system
Efferent vein of the dural sinus
Efferent vein
of the dural
sinuses
Trabecula
capillary plexus
of the infundibulum
Secondary
plexus of the
pituitary portal
efferent vein
of the dural sinus
lateral branch
and middle branch
of the inferior pituitary artery
Efferent veins of the dural sinuses

Fig. 72.2 Portal circulation of the pituitary gland.

Basophilic cells, with the same shape as acidophilic cells, make up 10% of the adenohypophysis. Four classes have been differentiated. It is generally accepted that basophil cells secrete thyroid-stimulating hormone or thyrotropic hormone (TSH), gonadotropins (FSH and LH), and adrenocorticotropic hormone (ACTH).

Chromophobic cells correspond to around 50% of the cells of the adenohypophysis. They are smaller than chromophilic (acidophilic and basophilic) cells.

Electron microscopy has revealed granules scattered in the cytoplasm in many cells, which correspond to stored hormones.

The name chromophobe cells dates back to the days when hematoxylin and eosin stains and light microscopes were available. Currently, with more refined techniques, secretory granules and small amounts of hormones are discovered in many of these cells. Adenomas of chromophobic cells have been observed in patients with manifestations of increased growth hormone (GH) or adrenocorticotropic hormone (ACTH), which demonstrates their functional activity, although some point out that chromophobic cells are not specific, but rather inactive or secretory exhausted cells without granules. In humans there is no well-developed pars intermedia and the cells are scattered throughout the anterior lobe. These pars intermedia cells can be assumed to look like chromophobe cells, which adds to the confusion.

The histology of the neurohypophysis is much simpler. The posterior lobe is made up of Bucy's pituicytes, thin, neural type and dispersed, between which there are nerve fibers and many small-caliber vascular ducts that anastomose.

PHYSIOLOGY OF THE PITUITARY

The physiological functions of the pituitary gland differ fundamentally according to the lobe, whether the anterior or posterior. Some consider the pituitary gland as a storage organ for hormones produced by the hypothalamic nuclei and which have reached it through neurosecretion mechanisms.

This explains why, in the absence of pituitary structural alterations, syndromes of pituitary hyperfunction (hyperpituitarism), hypofunction (hypopituitarism) and hypothalamic dysfunctions with pituitary repercussions occur in patients with hypothalamic lesions.

ANTERIOR PITUITARY PITUITARY (ADENOHYPOPHYSIS)

Diseases of the anterior pituitary manifest themselves in two ways: mechanically, when they are pituitary tumors that take up space and exert pressure on the neighboring structures and functionally, when there is excess or lack of secretion of any of the hormones produced by the gland or all of them.

In the last half of the 20th century, rapid advances occurred in physiological and biochemical knowledge of gland resection in animals and in humans, thanks to the work of many researchers. Methods were created for the extraction of increasingly pure hormones and for physiological, experimental and clinical studies; Knowledge was gained about the chemical structure of several hormones and the total synthesis of ACTH and melanocytostimulating peptides was carried out.

It was also possible to measure the concentration of hormones in the blood through radioimmunological methods.

plasma.

Subsequently, experimental and clinical research was concerned, above all, with the control of the nervous system over the function of the anterior pituitary gland, especially the release of hypothalamic hormones.

The microscopic anatomy of the anterior pituitary has been the subject of much discussion. The classic concept of acidophilic (eosinophilic and basophilic) and chromophobic secretory cells without function had to be modified according to subsequent data. Studies carried out with the electron microscope in the human adenohypophysis have made it possible to identify six or more different types of cells, probably responsible for each hormone.

Anteropituitary hormones

Six main hormones of the anterior pituitary are considered, whose biosynthesis, structure, function and control of their secretion have been defined quite accurately. They are:

1. Growth hormone, somatotropin or somatotropic hormone (STH or better, GH).
2. Thyrotropin or thyroid stimulating hormone (TSH).
3. Adrenocorticotropin or adrenocorticotropic hormone (ACTH).
4. Follicle-stimulating hormone or follicle-stimulating hormone (FSH).
5. Luteotropin or luteotropic hormone (LH).
6. Prolactin (PRL).

There are groups with similar structures. TSH, FSH and LH are glycoproteins, while GH and PRL are peptides.

It is also known that in the adenohypophysis there is another group with biochemical characteristics similar or related to ACTH, such as melanocyte-stimulating peptides, formerly called nelanocyte-stimulating hormone (MSH), lipotropin (LPH) and other endorphins, but they have not been identified with functions on specific target organs.

Each of the antepituitary hormones is regulated or released by hormones from the hypothalamus that stimulate the secretion of pituitary peptides.

Hypothalamic inhibitory factors also have an important regulatory influence. The control of prolactin secretion (PRL) is carried out by the prolactin inhibitory factor (PIF) and there are peptides that inhibit growth hormone (somatostatin) and melanocyte-stimulating peptides (formerly MSH).

Pituitary hormones act directly, like prolactin, or through secondary messengers.

The stimulating activity of growth hormone is probably carried out by an intermediate protein, somatomedin.

Gonadotropins, thyrotropin and ACTH stimulate the synthesis of hormones in their respective target organs, through cAMP.

Growth hormone or somatotropin (GH)

It is the substance that, secreted or administered in excessive quantities, produces gigantism and acromegaly in animals and humans.

Its absence causes difficulty in growth. It exerts its influence on somatic growth in man from the first stages of childhood. It has diffuse metabolic effects in young people and adults. Chemically, it is a single chain of peptides, with a molecular weight of 21,500. Approximately 500 mg are secreted per day. In pituitary dwarfism, basal plasma levels of growth hormone less than 1 mg/mL may be observed; These low figures do not allow the diagnosis to be established, because the normal limits are 0-5 mg/mL; Therefore, it is necessary to demonstrate that no hormone secretion reaction occurs to the hypoglycemic stimulus.

Its most notable action is anabolic. Several of its effects on cartilage are apparently carried out by an obligate intermediary, the "sulfation factor" called somatomedin, a protein with a molecular weight of 8,000 and which originates in the liver. Several types of somatomedin have been described.

Human growth hormone produces cellular hypertrophy or hyperplasia and thyroid hormones and insulin are necessary for an optimal growth reaction. It produces protein anabolism in the hypophysectomized subject or those suffering from hypopituitarism; It intervenes in the metabolism of water and electrolytes. Tissue protein and water are increased. There is moderate sodium retention and a positive overall calcium balance, although hypercalciuria may be present.

It also intensely modifies carbohydrate metabolism, making it diabetogenic;
many acromegalics have diabetes.

Serum growth hormone values increase rapidly due to hypoglycemia, fasting, and exercise.

Intravenous injection of amino acids, ACTH and glucagon, large doses of catecholamines and oral administration of L-dopa also stimulate the secretion of human growth hormone.

The intensity of secretion of this hormone is greater in hyperthyroidism and lower in hypothyroidism.

Apart from the obvious value of estimating serum concentrations of growth hormone for the diagnosis of hypopituitarism and acromegaly, the data mentioned above have shown that in adults it is not secreted at a uniform rate. The lability in the response, which is modified by metabolic events, suggests that it actively participates in the regulation of organic metabolism, day to day.

Thyrotropin, thyrotropic or thyroid-stimulating hormone (TSH)

Corrects thyroid atrophy after hypophysectomy and restores the gland's functional capacity. If administered in large doses to man or a hypophysectomized animal, it produces the morphological and secretory changes of hyperthyroidism, causes a rapid release of thyroxine and triiodothyronine in the gland, reduces its colloid content and decreases the amount of organic iodine.

Thyrotropin is a glycoprotein with a molecular weight of approximately 28,000.

The rapid release of thyroxine from follicles is due in part to the effect of TSH on a proteolytic enzyme that accelerates the separation of the hormone from thyroglobulin. TSH increases the ability of the thyroid to capture iodide and transform it and synthesize thyroxine.
These effects are reproduced by administering cAMP, so this and other data suggest that the nucleotide plays an important role in the effect of TSH on the gland.

Approximately 100 units of TSH are secreted per day. This figure does not vary with the different stimuli. Hypothalamic thyrotropin-releasing hormone appears to be the most important regulatory factor for TSH secretion. Thyroxine and triiodothyronine in turn have important effects on the hypothalamic and pituitary control of TSH secretion; For this reason, high concentrations are observed in cases of primary hypothyroidism and low concentrations in cases of spontaneous and iatrogenic hyperthyroidism.

Adrenocorticotropin or corticotropin (ACTH)

It stimulates the atrophied adrenal cortex and restores it to normal, allowing it to synthesize steroid hormones after hypophysectomy. It causes hypertrophy and hyperplasia of the adrenal glands, with a main effect on the fascicular and reticular areas.

Administration of large doses of ACTH produces adrenal enlargement and hyperemia. It stimulates the synthesis and release of cortisol, corticosterone and androgens, and a moderate and non-persistent increase in aldosterone.

The chemical nature of the hormone is known;
It is a polypeptide made up of 39 amino acids.
The hormone has been synthesized, with a molecular weight of 4,500. Daily production in humans is small, about 10 mg per day.

Administration causes a rapid increase in cortisol and its plasma concentration in humans.
This effect is useful for the diagnostic assessment of the adrenal corticosteroid response, as well as the biological and immunological assessment of ACTH in human serum.

The plasma concentration of ACTH follows its own daily rhythm, under the influence of the central nervous system (hypothalamic corticotropin-releasing hormone). The numbers tend to be higher in the early morning hours.

Plasma cortisol concentrations go hand in hand with this daily rhythm.

In man, the long-term effects of ACTH are the same as those produced by cortisol, cortisone and their synthetic counterparts.

ACTH regulates growth and steroid secretion from the adrenal cortex. This constantly changing influence is exerted by the action of the central nervous system that controls the anterior pituitary gland, and by a reciprocal relationship between the amounts of circulating cortisol and ACTH. In humans, noxious stimuli, an acute illness or an emotional disorder, cause an increased release of adrenal corticosteroids, probably by increasing the release of endogenous corticotropin. There are clinical tests to assess the adrenal response to ACTH and methods to estimate its pituitary release.

Follicle-stimulating hormone (FSH)

It is the pituitary hormone essential for the normal cyclic growth of the ovarian follicle and to restore follicular growth in the hypophysectomized female.

It is capable of stimulating spermatogenesis in the male, without significantly influencing the interstitial cells of the testicle. For FSH to have an optimal effect on the ovary and testicle, the existence of luteinizing hormone (LH) seems to be necessary.

The molecular weight is evaluated between 24,000 and 35, 000. It is a glycoprotein.

In infants and young children, gonadotropin concentrations are low; they increase before puberty. In women, during the preovulatory (follicle) and postovulatory (luteal) phases of the menstrual cycle, the figures are approximately the same as in men. In the latter, FSH and LH are secreted by arrhythmic pulsatile episodes (like ACTH) during sleep.

Pituitary control of the secretion of both hormones is established at the hypothalamic level, by positive feedback control.

During the first part of the follicular phase, the plasma concentration of FSH increases, starting from the minimum levels during menstruation; then, as estrogen secretion increases, during the middle part of the menstrual cycle, it decreases. In this phase, the plasma concentration of LH increases progressively, until reaching the middle part of said cycle. Immediately following peak levels of estrogen (which is thought to stimulate the release of the gonadotropin LH), peak secretion of FSH and LH occurs. The average secreted amounts of FSH and LH are 15 and 30 mg per day, respectively, and increase 10-15 times during the middle phase of the menstrual cycle, with ovulation.

The plasma levels of these hormones immediately decrease in the luteal phase. With high progesterone values, the FSH concentration level reaches its lowest values. LH rises slightly as estrogen and progesterone decrease, before menstruation.

If they are not counteracted by the effect of negative feedback of gonadal hormones, plasma and urinary FSH levels are high, as occurs in menopausal and ovariectomized women. The negative feedback is due in part to estrogen inhibition of the hypothalamic hormone that releases gonadotropin. In men, the inhibitory testicular hormone is possibly produced by the seminiferous tube, since testosterone suppresses FSH secretion in a mild manner.

Luteinizing hormone or interstitial cell-stimulating hormone (LH)

Luteinizing hormone is essential in ovulation, the formation of the corpus luteum and its stimulation to secrete steroid hormones. For its effect to be effective, it is necessary that FSH has previously acted on the follicle. It is accepted that human LH is the luteotropic hormone.

This hormone stimulates the growth and secretory activity of the interstitial cells of the testicle, enough to correct the testicular endocrine defect of the hypophysectomy, causing secretion of androgens and preservation of the secondary sexual organs.

The steroidogenic effect of LH on the corpus luteum and Leydig cells is carried out through cAMP. Spermatogenesis requires the synergistic effect of FSH.

LH is a glycoprotein whose molecular weight is 28,000. Its hormonal specificity is produced by the chemical characteristics of the B subunit.

The chorionic gonadotropin, which is more easily obtained, has LH-type effects and is the one used clinically instead of the hormone.

Testosterone, estrogens, and progestins inhibit LH secretion, in part, through their effect on the production of hypothalamic releasing hormone LH and FSH. In men, testosterone decreases plasma LH values. In women, estrogen and progesterone preparations (in antiovulatory drugs) eliminate the increase in LH in the middle phase of the menstrual cycle and reduce the secretion of this hormone in postmenopausal patients. Conversely, LH levels are high in castrated men and in women during postmenopause.

It appears that the concentration of circulating estrogens in the follicular phase of the menstrual cycle stimulates the release of LH.

Prolactin or lactogenic hormone (PRL former LTH)

When prolactin was discovered, it was shown that it was a different hormone from human growth hormone, despite having many chemical and biological characteristics in common.

The data that support their differences are the following: the human pituitary gland during pregnancy and lactation, and in cases of galactorrhea, contains many acidophilic granules that do not resemble those that secrete growth hormone; Patients with galactorrhea do not have high plasma concentrations of growth hormone and this does not increase during lactation; Normal postpartum lactation is achieved in mothers who suffer only from growth hormone deficiency; Pituitary tumors associated with galactorrhea contain significant levels of prolactin activity, which cannot be neutralized by anti-growth hormone; and, finally, it has been observed that prolactin is immunologically different from growth hormone.

In women, prolactin controls the growth, development and formation of milk in the breast. The physiological role of prolactin in men is not well known.

The hormone is a polypeptide chain with a molecular weight of 23,000. It is chemically similar to growth hormone and human placental lactogen.

Prolactin exerts more than 20 physiological effects, most of them related to reproduction. It has effect growth-stimulating cough, similar, but less intense, to growth hormone and is secreted in periodic episodes. Plasma levels are lower during waking hours, increase 60-90 minutes after beginning to sleep and reach maximum concentrations from 5:00 to 7:00 in the morning; They do not vary from day to day during the normal menstrual cycle, but they increase progressively during pregnancy and reach a noticeable increase during lactation.

Various physiological stimuli produce increased secretion of prolactin, such as: breast feeding in women after childbirth, stimulation of the non-lactating breast, intense physical exercise, sexual relations in women (especially during orgasm) , hypoglycemia and surgical trauma. The concentration of prolactin is also increased after administering ERT, estrogens, arginine, phenothiazines and other tranquilizers (which sometimes cause galactorrhea) and reserpine. Prolactin concentration is decreased by prolonged fasting, L-dopa, nicotine, and ergot alkaloids.

Melanocyte-stimulating peptides (formerly MSH)

They produce darkening (Addisonian pigmentation) of human skin.

Two MSH peptides have been discovered in pituitary tissue.

In animals, the smallest and most potent molecule (LMSH) is found in the pituitary gland of pigs, cows, monkeys, and possibly the human neurohypophysis. BMSH, both in animals and humans, has common properties with ACTH; However, in humans there is no MSH itself, but rather peptides that share properties similar to ACTH, in the stimulation of melanocytes and which we will sometimes continue to call MSH, now that clarification has been made.

This explains, perhaps, the common physiological properties of its molecules: the slight skin pigmentation effect caused by ACTH; the mobilization of fat reserves by MSH and ACTH; the mild adrenal-stimulating activity of MSH and the secretion of MSH regulated by the same factors that control the release of ACTH.

The functional importance of BMSH in humans is unknown. Its levels are normal in cases of albinism and in serious diseases characterized by increased dermal pigmentation.

Control of anterior pituitary secretion

Although the anterior pituitary gland (adenohypophysis) lacks direct innervation, the secretion of each of its hormones is under control of the CNS, thanks to secretory neurons located in the ventral hypothalamus; The function of the pituitary gland and, at the same time, that of its white glands, respond to changes in the external and internal environment.

The neurohormonal connections of the anterior pituitary are also important for regulating by feedback a certain number of hormones: cortisol, gonadal steroids, thyroxine, prolactin, as part of the integrated mechanism by which metabolic and behavioral adaptation to the external environment is achieved (Fig. 72.3).

In recent years, as the importance of neural factors in the control of pituitary secretion has become increasingly evident, what is known as neuroendocrinology was born, which deals with the interaction of nervous and hormonal factors in endocrinometabolic control systems.

Hypothalamic neurosecretion: hypophysiotropic neurons

The pituitary gland and hypothalamus have a close anatomical relationship. This relationship has embryological and functional significance.

The cells of origin of this neurosecretion are located in the paraventricular and supraoptic nuclei of the hypothalamus. Its nerve fibers continue in the form of non-myelinated axons through the neural stem and end in the nervous lobe of the pituitary gland. Like neurons in other parts of the body, they are electrically excitable, conduct action potentials, and respond to neurotransmitters.

They are neurosecretory cells, since they synthesize hormonal substances that, once released, pass into the blood vessels and act in distant areas. The hormones of the neurohypophysis are synthesized mainly in the cell bodies located in the hypothalamus and pass in the form of granules (associated with a carrier protein substance) to the nerve endings of the neural lobe (median eminence), where they are stored.

Neurosecretory neurons thus manifest the properties of nerve and gland. These cells have been called neuroendocrine transducts by Wartman.

Cells of this type constitute the main effector junctions between the endocrine glands and the nervous system.

The secretions of the neurohypophysis (vasopressin and oxytocin) do not exert local action on the anterior pituitary, despite its proximity, but instead pass into the general circulation and modify distant tissues: kidney, breast, uterus, etc.

The phylogenetically most primitive neuroendocrine " transducer" is located in the median eminence of the hypothalamus. It is accepted that the nerve endings at this level contain hypophysiotropic hormones, also called release factors, which reach the anterior pituitary following the specialized system of capillaries and veins (portohypophyseal system), as has been pointed out.

The cells of origin of the median eminence are gathered around the medial-basal hypothalamus in the so-called hypophysiotropic zone. They resemble neurohypophysial neurons in being electrically active and excitable by drugs; They respond to a wide variety of nervous impulses that come from other parts of the brain and also to feedback hormonal stimuli. Direct electrical stimulation of tuberohypophysial neurons or their reflex activation by emotional stimuli activates the anterior pituitary by releasing hypophysiotropic hormones.

Figure 71.2 of the previous chapter shows the anatomical relationships and functional control elements of the hypothalamic-pituitary unit. Two general types of neurons come into operation:

The first type corresponds to the neurons that produce release hormones and to which the so-called peptidergic neurons belong; of these, some end in the median eminence and others in the pituitary stalk, both in relation to the capillary plexus of the portohypophyseal vessels. Peptidergic neurons are neurosecretory and transform neural information into endocrine; They are neuroendocrine transducers. Its function is similar to that of the classic pituitary supraoptic neuron, which is responsible for the secretion of vasopressin.

The other type is made up of neurons that form the junction between the rest of the brain and the peptidergic neurons. Neurons of this type are largely monoaminergic and are believed to terminate on the cell body of peptidergic neurons (ordinary form) or at the end of the axon (so-called axoaxonic).

This scheme of the regulation of the anterior pituitary represents the theory of chemotransmission of the portal vessels, already studied.

GENERAL CONSIDERATIONS OF PITUITARY SYNDROMES

When doing the clinical study of pituitary syndromes, we must remember that primary lesions of this gland can produce two types of symptoms: hormonal and mechanical.

As the pituitary gland is the source of many tropic hormones, injuries that increase or decrease their production inevitably cause effects on the endocrine glands that depend on them.

Hyperfunctioning tumors of the pituitary gland produce excessive activity in the thyroid, adrenals, and gonads.

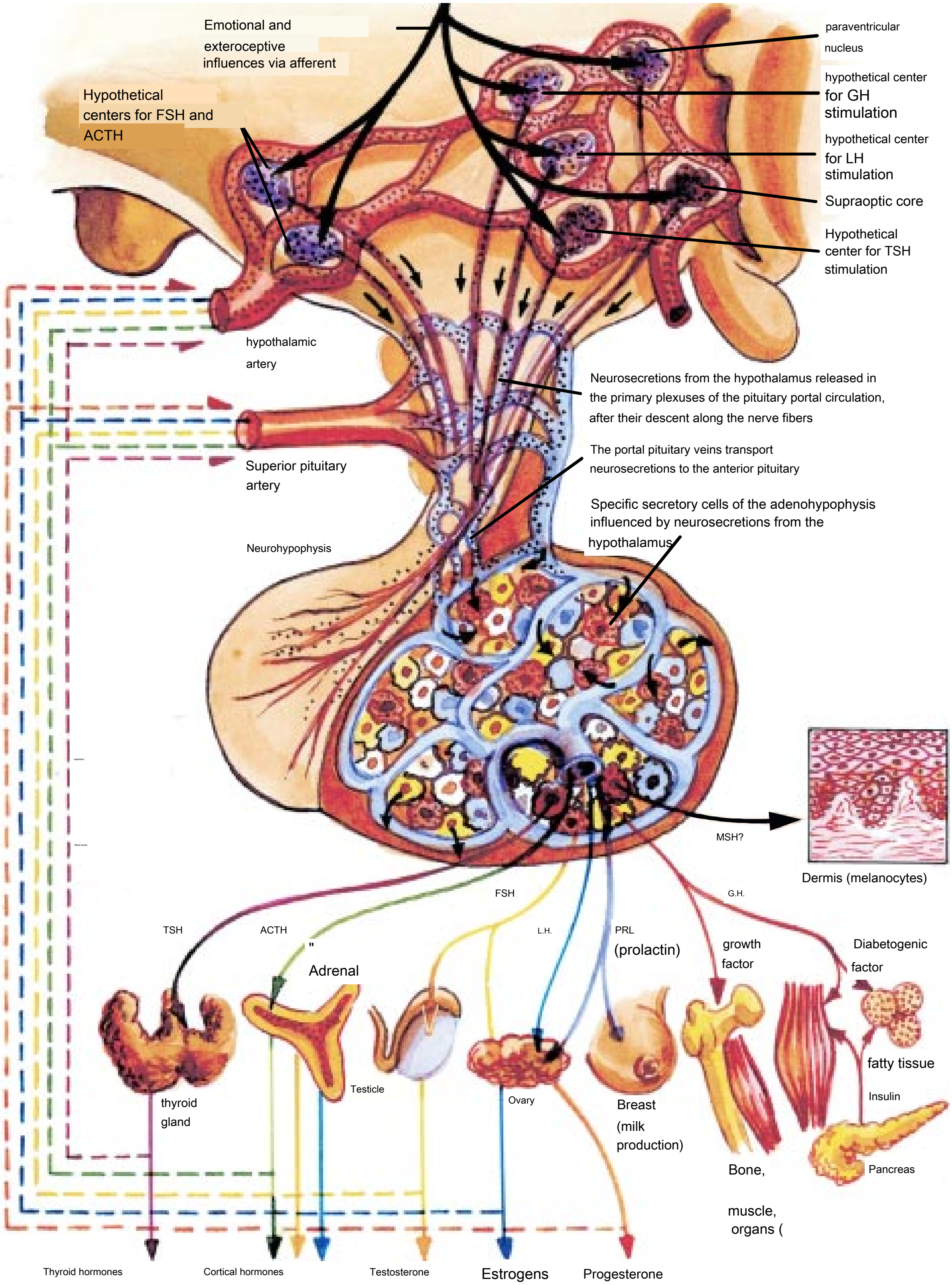

Emotional and
exteroceptive
influences via afferent
paraventricular
nucleus
Hypothetical
centers for FSH and
ACTH
hypothetical center
for GH
stimulation
hypothetical center
for LH
stimulation
Supraoptic core
Hypothetical
center for TSH
stimulation
hypothalamic
artery
Neurosecretions from the hypothalamus released in
the primary plexuses of the pituitary portal circulation,
after their descent along the nerve fibers
Superior pituitary
artery
The portal pituitary veins transport
neurosecretions to the anterior pituitary
Specific secretory cells of the adenohypophysis
influenced by neurosecretions from the
hypothalamus
Neurohypophysis
MSH?
Dermis (melanocytes)
FSH
G.H.
TSH
ACTH
L.H.
PRL
(prolactin)
growth
factor
Diabetogenic
factor
"
Adrenal
fatty tissue
Testicle
Ovary
Breast
(milk
production)
Insulin
thyroid
gland
Bone,
muscle,
organs (
Pancreas
Thyroid hormones
Cortical hormones
Testosterone
Estrogens
Progesterone

Fig. 72.3 Control of pituitary secretion and hormones.

Lesions that cause destruction of the pituitary gland with the disappearance of tropic hormones are manifested by regressive changes in these glands and their effector organs.

Lesions that grow and expand produce their effects by virtue of the increase in volume; The effect can be localized, such as the local destruction of the sella turcica. In more advanced cases, compression of adjacent organs (floor of the third ventricle, optic chiasm, hypothalamus) causes symptoms.

Compression of the optic chiasm produces a bitemporal hemianopsia.

Large pituitary tumors produce a general increase in intracranial pressure and can, like any brain tumor, cause headaches, vomiting and other symptoms that reflect the increased pressure of the cerebrospinal fluid (CSF).

Although both aspects are important, the main clinical characteristics usually refer to the functional alterations that originate from hormonal imbalance.

In an analysis of pituitary tumors, 40% presented local symptoms, 57% endocrine symptoms and the remaining 3% of the tumors were discovered randomly, asymptomatic.

INJURIES AFFECTING THE PITUITARY GLAND

Pituitary function can be disrupted by various pathological states of the gland itself and thus, we see that it can be decreased by hypophysectomy or by radiation sometimes used in patients with breast cancer. Symptoms of pituitary insufficiency only become apparent when 70% or more of the anterior lobe has been destroyed.

Acute or chronic inflammations and rare acute bacterial infections result from bacteremic seeding or direct spread of infection from the leptomeninges, dural sinuses, or bone.

Chronic inflammatory states are observed in:
generalized sarcoidosis, miliary tuberculosis and congenital syphilis. More rarely, toxoplasmosis and actinomycosis, like other fungal infections, affect the pituitary gland.

There are infiltrations, with deposits of amyloid substance, in generalized amyloidosis; In Hand-Schüller-Christian disease (infiltration by lipophages) the bone is damaged and the pituitary gland is infiltrated, especially the posterior lobe; In glucopolysaccharidoses (Hunter and Hurler syndrome), the cells of the adenohypophysis may present accumulation of mucopolysaccharides.

As vascular lesions, microscopic foci of necrosis produced by injuries are quite common. small vascular lesions: extensive infarction of the pituitary gland may be a consequence of generalized atherosclerosis, affecting the internal carotid or its pituitary branches, or of thrombosis of the cavernous sinus; Another mechanism is embolic obstruction of the vessels that supply the pituitary gland;
Much more common is massive ischemic necrosis of the anterior lobe, secondary to shock.

In the past, this condition was more common in women who had severe hemorrhage after childbirth and the name Sheehan' s postpartum necrosis was given to the relationship between postpartum and pituitary damage.

Today, similar cases are observed in non-pregnant women and men, so it is better to call the condition Sheehan's syndrome, which can be caused by septic shock, burns, or massive hemorrhage from any cause. The damage is considered to result from vascular insufficiency or concomitant thrombosis, due to a sudden drop in blood pressure. It is proposed that disseminated intravascular coagulation constitutes the intimate mechanism of pituitary necrosis in these circumstances (McKay et al.).

Tumors are the most frequently observed anatomopathological lesions in the pituitary gland (in addition to Sheehan syndrome),

Adenomas are the most common benign neoplasms and can constitute 60% of pituitary tumors. With the advent of computed axial tomography (CT) and magnetic resonance imaging (MRI), adenomas have been divided into microadenomas (< 10 mm) and macroadenomas (> 10 mm). They can be eufunctioning or, better, non-functioning, which constitute 50% of all adenomas, or they can generally cause pituitary hyperfunction.

Adenomas formed by somatotropin cells cause gigantism and acromegaly; those with corticotropic cells, Cushing's syndrome and prolactinomas, formed by lactotropic cells (PRL producers), which constitute the most common group of adenomas.
originate., when they are hyperfunctioning: galactorrhea, amenorrhea and female infertility, as well as sexual dysfunction and galactorrhea in men. There are adenomas with mixed cell populations and hormonal production can be varied.

In second place, in order of frequency, are suprasellar tumors, such as craniopharyngioma, Rathke's pouch tumor, and adamantinoma. Other benign tumors are rarer: fibromas, hemangiomas, teratomas, cholesteatomas.

Primary malignancies are also rare;
Sometimes they develop in adenomas or preexisting craniopharyngiomas.

Secondary malignant tumors, metastatic from the mammary gland, lung or thyroid, are also not frequent.

As we have studied, adrenal, thyroid and gonadal hormones produce inhibition, by feedback on the corresponding secretory function, in the anterior pituitary. The modifications (increase or decrease) of the hormonal concentrations produced by the endocrine effector organs (target or target organs) have secondary opposite actions on the adenohypophysis and cause the syndromes of adrenal corticosteroid, thyroid or gonadal hyperfunction or hypofunction.

ANTERIOR LOBE SYNDROMES

HYPERPITUITARISM SYNDROME

Concept

In fact, hyperpituitarism should be called any syndrome that results from the hypersecretion of any of the tropic hormones or pituitary tropins.

Although these conditions actually appear quite frequently, they are still not well known to date.

The multiplicity of cells with different immunohistochemical characteristics corresponds to the secretion of different stimulating hormones, which can result in interference in the production of some by others. In these circumstances, hyperfunction or hormonal hyperstimulation syndromes can take on different aspects:

1. Simple: they only affect one endocrine function (gonadal, thyroid, adrenal corticosteroid, etc.).
2. Complex: they affect more than one endocrine function.
3. Dissociated: with hyperstimulation on one endocrine axis and hypostimulation on another.

Classification

Hyperpituitarism or hyperstimulation syndromes can be:

Organic lesional hyperstimulation syndromes.

- Reactive or functional hyperstimulation syndromes.

Injury or organic hyperstimulation syndromes

They are rare and frequently depend on a hypersecreting adenoma of somatotropin, corticotropin, or lactotropin cells. They generally constitute complex, multi-endocrine syndromes associated with pituitary tumor signs.

Functional reactional hyperstimulation syndromes

They are, on the contrary, very common and depend on an abnormal hyperexcitability of the hypothalamic direction of the pituitary gland.

Abnormal hyperexcitation is sometimes the consequence of a lesion of the hypothalamus, due to basal meningoencephalitis or brain tumor.

This immense stimulus, exerted on abnormally irritated hypothalamic centers, can be produced by massive hormonal deficiency at the level of tissue receptors and by interference from too intense stimuli of external origin, whether physical, sensory or psychogenic.

In both cases, the hypothalamus is stimulated, which reacts with signs of nervous hyperexcitability, which affects pituitary secretion and gives rise to the development of a hyperstimulin syndrome.

The clinical appearance of hyperstimulation syndromes can be very varied:

1. Hormonal hypersecretion can provoke, in turn, a similar response at the level of the corresponding peripheral gland and thereby give rise to the appearance of total hyperhormonal syndromes, which include:

   a) For corticotropin: certain chronic functional hypercorticisms and Cushing's disease (see adrenals).

   b) For thyrotropin: classic Basedow disease.

   c) For gonadotropins: the so-called hyperfolliculinic syndromes, the hyperhormonal syndromes of premenopause, certain hyperhormonal syndromes of puberty, etc.

   d) For somatotropin: certain acromegaloid states, certain so-called pituitary diabetes and, at least in part, certain cancers, adaptation diseases, etc.

2. On other occasions, hormonal hypersecretion is exerted on peripheral glands incapable of responding, either due to an organic alteration (sclerosis, involution, infection) or due to a pathological deficit of receptivity. In these conditions, an isolated hyperfunction syndrome appears, which is accompanied by hypofunction of the corresponding peripheral gland. Such dissociated hyperhormonal syndromes are not perfectly described yet.

   However, the following are known:

   a) For corticotropin: the hypercorticism (melanoderma) syndromes of Addison's disease.

b) For thyrotropin: hyperpituitary forms of Basedow's disease, especially malignant edematous exophthalmias and hyperthyrostimulin syndromes observed in the course of certain thyroid insufficiencies, after improper administration of synthetic antithyroid extracts.

c) For gonadotropins: hyperfolliculostimulin syndromes of menopause, both spontaneous and surgical.

d) For prolactin: galactorrhea, amenorrhea and infertility.

Symptoms and complementary tests

Hyperstimulation syndromes are extraordinarily complex and require extensive careful study. Below, the symptomatic characteristics that are triggered by the stimulation of the different glands and the laboratory evaluations that are within the reach of every practical doctor will be briefly explained; We will only be more extensive regarding the hypersecretion of somatotropin, as it is the one that manifests more defined symptoms and without the intervention of any intermediate peripheral gland.

Adrenal gland

The corticotropin that acts on this gland is ACTH. It is estimated that the most characteristic action is the condition called global adrenal hypercorticism (see Adrenal Cushing's).

ACTH, in its hyperstimulinic action on the adrenal gland, causes the appearance of the following clinical symptoms: faciotroncular obesity (buffalo), abdominal purple streaks or streaks, hirsutism, high blood pressure and painful osteoporosis.

Together with the previous symptoms, the following alterations may be indicated in complementary tests: polyglobulia, lymphopenia, water and sodium retention, hyperglycemia and glucosuria, provoked hyperglycemia (altered diabetic type), elevation of urinary content in 17-ketosteroids and 17-oxycorticosteroids.

This condition is called global adrenal hypercorticism or Cushing's disease.

thyroid gland

The thyroid-stimulating hormone is TSH and it causes, by acting on the thyroid gland, the appearance of the following clinical symptoms: hyperplasia of the gland (goiter), tachycardia, weight loss, psychomnotor hyperactivity, increased sensitivity to heat, tremors, etc.

Along with the previous symptoms, alterations may be required in the following complementary tests: low blood cholesterol, elevated protein iodine level (PBI), elevated radioactive iodine uptake (15'), elevated serum thyroxine (T4) and elevated serum triiodothyronine (T3); These last two are the most important exams.

The syndrome whose characteristics we have just mentioned is called hyperthyroidism or thyrotoxicosis.

Sex glands (ovaries and testicles)

Due to its own characteristics, according to the sex of the individual, we will study it separately.

In women, the stimulating hormones that act on these glands are several: FSH, LH and PRL;

The action of the latter is selective on the breasts.

The hormones FSH and LH act causing striking symptoms, such as: the hot flashes of menopausal syndrome. PRL produces premenstrual congestion and its excessive elevation, galactorrhea-amenorrhea, infertility and hirsutism.

In men, it is estimated that the FSH and LH hormones, when elevated, act by provoking, as their most characteristic action, erotic tendencies of a pituitary nature, which require being well defined in terms of their etiology, which is usually, in general terms, quite difficult, since the possibility that they are psychoneurotic manifestations, which are usually more frequent, must be ruled out. These hormones, in their hyperstimulating action on the testicles, would cause the following symptoms: somatic changes, decreased height, short neck, wide and reddish facies and heightened libido.

Acromegaly

Concept

It is a clinical syndrome caused by the pathological increase in the growth hormone, somatotropin (GH), which occurs in adults over 30 years of age and is characterized by an extraordinary development of the bones and soft tissues, more distal to the body.

Syndromogenesis or pathophysiology

The increased concentration of growth hormone in adults stimulates the growth of connective tissue, cartilage and bones. This growth produces an increase in volume of the skull, hands and feet, and macrosplannia. In general, there is no increase in height, since by definition it appears after the endochondral formation of bone has been completed, with consolidation of the epiphysis.

Syndromography or positive diagnosis

Clinical picture

The onset is usually insidious and the first symptoms are purely subjective: diffuse pain, weakness and muscle weakness.

On other occasions, it is the alterations typical of the period of the disease state that draw attention, especially in an evolutionary way, over the years (fig. 72.4).

The morphological manifestations Are:

1. Weight gain.
2. Normal or slightly increased size.
3. Curvature of the dorsal column due to anterior widening of the vertebral bodies and osteocartilaginous confluence; In addition, there is bone apposition and cartilaginous growth of the menisci.
4. Alterations of the face: prognathism of the lower jaw, increase in the superciliary and zygomatic arches, enlargement of the paranasal sinuses, marked occipital protuberance. As a result of bone development, the teeth separate, especially those of the lower arch (diastema). Overall, there is a predominance of the facial mass over the skull, with concomitant alterations of the cartilaginous formations and soft tissues: the cartilages of the nose grow, which widens this organ; 1The ears are also affected, increasing in size; The lips increase in thickness and the tongue bulges, becomes large (macroglossia), with deep striations (scrotal tongue).
5. The skin usually increases in thickness, has a callous appearance, wide pores, with abundant sebum and sweat secretion. These soft tissue alterations force the skin of the cheeks to fold, which accentuates the facial features and gives the patient a characteristic grotesque or bestial appearance (see also Figure 24.14, volume 1, Chapter 24).
6. In a similar way, the extremities grow abnormally, particularly the hands and feet, which acquire a rough appearance: spade-shaped hands, with sausage-shaped fingers, due to an increase in the soft parts: periosteal proliferations on the ends of the fingers; The feet also grow, especially in width.
7. In addition, splanchnomegaly is usually observed, that is, a great increase in visceral size, including the heart; A marked development of the external genitalia is observed, in opposition to the more or less marked atrophy of the ovaries and testicles.

Between endocrine and metabolic manifestations have:

1. The thyroid may be enlarged and there may be temporary signs of hyperthyroidism; In advanced periods, hypothyroidism is common.
2. The adrenal cortex may be hyperplastic at first and may cause hirsutism (later the cortex becomes exhausted).
3. The gonads undergo the variations already mentioned.
4. Overt clinical or chemical diabetes occurs due to the diabetogenic action of growth hormone.

Let's look at the tumor manifestations:

They occur in the very advanced stages of the disease. Its early appearance raises the suspicion of the possibility of carcinoma.

These manifestations are caused by the compression exerted by the tumor on neighboring structures or by the growth of the pituitary gland by overflowing the sella turcica through its weakest part, the diaphragm of the sella or tentorium of the pituitary gland. The most common symptoms are summarized below:

1. Headache, a frequent and annoying symptom.
2. Vision disorders: decreased visual acuity or total blindness and bitemporal hemianopia.
3. The symptoms of hypothalamic compression are less frequent: epilepsy, narcolepsy, obesity, hypothermia, hyperthermia and diabetes insipidus.
4. Signs of intracranial hypertension will be suspected when they appear: vomiting, bradycardia, papillary edema and hypertension.

Complementary exams

1. Increased serum inorganic phosphorus: more than 4 mg%.
2. Increase in urinary 17-ketosteroids.
3. Hyperglycemia.
4. Positive nitrogen balance (unlike Cushing's).
5. Normal or low urinary gonadotropins.
6. Hypercalcemia, in some cases; hypercalciuria is common. These alterations disappear with appropriate treatment. In other cases, hypercalcemia persists and may be an expression of associated hyperparathyroidism, as part of the multiple endocrine adenomatosis syndrome.
7. Radiological alterations: there are pathological alterations of the sella turcica, such as its enlargement with rupture of the floor of the sella and the anterior and posterior clinoids. Sometimes you can only see

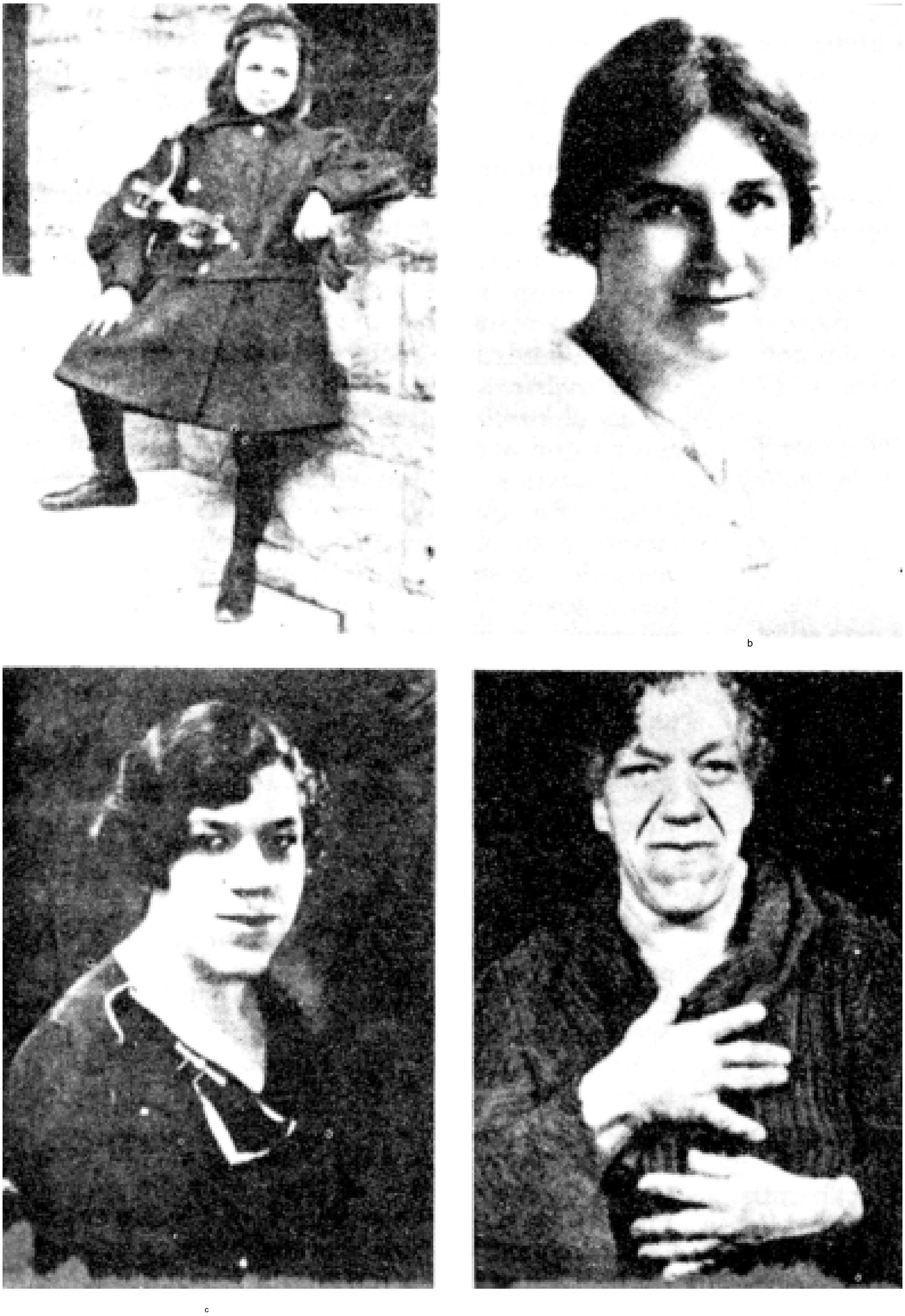


Fig. 72.4 The progression of acromegaly is illustrated in the following photos: a, age 9 years (normal); b, age 16 years (with possible early coarseness of features); c, age 33 years (well-established acromegaly); d, age 52 years (final stage of acromegaly with gross disfigurement).

adenomas by computed axial tomography (CT) and magnetic resonance imaging (MRI) where hyperdense images are visualized.

8. Radioimmunological study of growth hormone. It is performed as a systematic procedure in specialized centers; It is the most direct method to detect pituitary disorders and acromegaly. Most untreated acromegalic patients have elevated growth hormone concentrations; After hypophysectomy or pituitary irradiation, these concentrations decrease to normal values. The determination of somatomedin C is also carried out, which is elevated.

9. There are other tests such as: glucose administration and dosing, for 3 hours, of growth hormone and blood glucose; In healthy subjects, the plasma concentration of growth hormone decreases noticeably; Many untreated acromegaly patients do not present this decrease.

Etiology

The cause is generally tumorous and is due to an adenoma of acidophilic cells of the pituitary gland (90% of cases), which enlarges and breaks the physiological limits of its continent, the sella turcica. Malignant lesions are rare.

In other cases, acromegaly evolves without a tumor (acromegalic disease); It is a relatively rare disease and affects both sexes, although it is somewhat more common in men.

Pituitary gigantism

Concept

Unlike acromegaly, which generally only occurs in adults (although cases have been described in young children), the so-called pituitary gigantism is attributed to the hyperfunction of the prehypophysis that occurs in youth, before the cartilage connecting the diaphyses with the epiphyses ossifies, that is, before the age of 20. It is usually observed in males.

Such cases form a not small contingent of the giants exhibited in circuses. The majority die before the age of 30; At autopsy, notable splanchnomegaly appears: heart, liver, spleen, kidneys and other internal organs, as does macrosplanchnia in acromegalic patients.

Syndromogenesis or physiopathology

It is identical to acromegaly, but the clinical appearance is different. A marked increase in growth hormone in the child determines an excessive, but generally proportionate, height due to bone growth. simultaneous epiphyseal. These patients do not present the distal deformities so characteristic of acromegaly.

Syndromography or positive diagnosis

clinical picture

At first, only the excess height of the child usually attracts attention, so in the face of any extreme variation in height it is advisable to specify the normal values at different ages, comparing the height tables, in the certainty that any growth that disproportionately exceeds that which corresponds to that age and sex forces, even in the absence of symptoms, to think about gigantism and look for an increase in the concentration of growth hormone in the plasma, by the radioimmunological method.

In the majority of patients, both gigantism and acromegaly, the evolution is insidious, and often, these diseases are not noticed by the patient or the family for years, since the progressive and slow changes in structure and appearance appear subtly.

The morphological manifestations are:

1. Excessive development of the skeleton, sometimes at the expense of arms and legs.
2. Vicious attitudes: kyphosis, flat feet and genu valgum.
3. Muscle weakness and thinness.
4. Easy susceptibility to infections.
5. Occasionally, facial hyperprognathism, mild exophthalmia and dental diastema.

The endocrine manifestations are:

1. Along with excess height, gonadal hypopituitarism is observed: psychological infantilism and delayed genital development.
2. Fleeting hyperthyroidism (sometimes).

Complementary exams

1. Bone x-rays: osteoporosis.
2. Elevated blood phosphorus.
3. Elevated growth hormone or GH, with pathological PTG test, without braking or inhibition of plasma GH levels.
4. Elevation of somatomedin C.

The interrelationship of the pituitary gland with the other endocrine glands is the reason why its alterations cause very noticeable variations in the appearance and symptomatology of these cases. For this reason, it is necessary to evaluate different types of gigantism.

It is justified to question the pituitary etiology when:

1. There are no signs of acromegaly.
2. The sella turcica is not enlarged.
3. There is many family history of extremely tall stature and, consequently, constitutional gigantism.

In harmonic, non-endocrine, constitutional, primordial or genotypic (inherited) gigantism, a simple increase in height is seen, proportional in all segments, and from whose study it follows that:

1. The subject's skeleton is made up of long, solid bones.
2. Their sexual and psychological characteristics are normal; preserves sexual power, libido and intelligence.
3. Laboratory tests are normal.

Hypogonadal or eunuchoid gigantism presents:

1. A skeleton made up of long, immature bones.
2. Predominance of length of the lower limbs over the upper ones and the breaststroke is greater than the height.
3. Sexual and psychological characteristics: slow ideation, sexual impotence, frigidity.
4. Laboratory tests indicate: decreased or absent urinary gonadotropins and very low 17-ketosteroids.

Acromegaloid gigantism presents:

1. Skeleton made up of wide, dense bones.
2. Sexual and psychological characteristics: great sexual development and sometimes slow ideation, respectively.
3. Laboratory tests indicate: phosphoremia of 4-6 mg, decreased urinary gonadotropins, and elevated 17-ketosteroids.

Etiology

It is the same as that indicated in acromegaly.

HYPOPITUITARISM SYNDROME: PITUITARY INSUFFICIENCY

Concept

This is the name given to the clinical picture resulting from the destruction of the anterior pituitary gland with secondary atrophy of the gonads, thyroid and adrenal cortex.

It is also known as panhypopituitarism or panhypophysism, which indicates the total absence of pituitary secretions.

Simmonds disease is another synonym. When the cause is puerperal necrosis of the gland, it is known as Sheehan syndrome.

Syndromogenesis or pathophysiology

On a pathophysiological level, antepituitary insufficiency does not differ from other endocrine insufficiency syndromes; In it there is an extinction of the physiological activities of the gland.

Taking into account the numerous physiological activities of the antehypophysis, the deficits will be numerous:

Direct signs of hormonal insufficiency: nanism in the child due to insufficient growth hormone.

Indirect signs of hormonal insufficiency: placement of three peripheral glands at rest:

1. Adrenocortical insufficiency due to suppression of corticotropic function.
2. Thyroid insufficiency due to suppression of thyrotropic function.
3. Gonadal insufficiency due to suppression of gonadotropins.

4. Alteration of various metabolisms: for example, hypoglycemia due to ACTH and GH deficiency.
5. Complex deficiency disorders; Let us point out three examples:

   a) Hydroelectrolyte metabolism disorders that depend on cortisone and aminolocorticoids; They are often partly masked by the associated deficit of antidiuretic function, which is due to the same etiology.

   b) Hypothalamic-pituitary disconnection syndromes, where the same destructive lesion will produce a deficit (amenorrhea due to suppression of activating factors of gonadotropic cells) and a secretory acceleration (galactorrhea due to suppression of the inhibitory factor of prolactin cells). This is how amenorrhea-galactorrhea syndrome develops.

   c) Finally, certain pituitary adenomas are responsible for the hypersecretion of a tropin, but at the same time they act, due to their volume, as an expansive process and due to this fact they give rise to compression, responsible for pituitaryism in the other sectors.

Syndromography or positive diagnosis

Clinical picture

A clinical history of postpartum hemorrhage, symptoms and signs of intracranial tumor with chiasmatic compression, or signs of multiple glandular deficiencies may be collected.

The nature and volume of the lesion explain the rapid onset of pituitary insufficiency.

Anterior lobe deficiency in adults (Fig. 72.5) corresponds to Simmonds disease (pituitary cachexia), which occurs more frequently in women. Although anorexia is very evident in some patients, cachexia is rare. It only appears as a terminal event in the untreated patient and in very advanced cases of anorexia nervosa (see Fig. 24.11, volume 1, Chapter 24). When it appears after hemorrhage during childbirth, it is called Sheehan syndrome (Fig. 72.6).

After an obstetric accident, the symptoms set in imperceptibly after months or years. Sometimes it is the inability to breastfeed the newborn, other times it is the loss of pubic and axillary hair. Menses do not recover or are scarce and transitory, followed by amenorrhea. Libido is lost. Later, symptoms of thyroid and adrenal cortico insufficiency appear, although not always in the established order.

When puerperal necrosis of the pituitary gland is extensive and severe, symptoms such as diabetes insipidus and hypoglycemia appear. In the most severe cases, within a few weeks the patient may die from acute adrenal

In general, the patient is weak, fatigues easily, and is indifferent to her personal hygiene and her home. There may be personality changes with symptoms of depression and frank psychosis. Sometimes it presents with emaciation of the body, early senility and dry and wrinkled skin, producing the configuration of the "goose's foot" with wrinkles around the mouth and along the cheeks. The manifestations of hypothyroidism are accentuated, such as marked intolerance to cold, constipation and loss of the external third of the eyebrows. The breasts usually suffer from atrophy, although in rare cases they may be preserved. There is atrophy of the genital organs.

The typical clinical picture is very evident in the late phase (15-20 years after the obstetric accident). The main cause of death is adrenal corticosteroid insufficiency.

Anterior lobe deficiency in men is suspected by the disappearance of libido and sexual potency (previously normal) with or without genital atrophy, in addition to the symptoms indicated for women (see Fig. 72.5).

In both, blood pressure is low, pulse slow and weak. Sometimes there is a picture of collapse preceded by clinical manifestations of nausea, vomiting, high fever and hypotension (adrenal crisis).

Pituitary coma with hypotension, hypothermia, and hypoglycemia may occur gradually (without apparent cause) or rapidly, in response to a minor illness.

Intrasellar tumors that increase in volume, such as chromophobe cell adenoma, may also have an insidious onset. In these cases, death generally occurs due to mechanical compressive disorders.

The interrelationship of the pituitary gland with the other endocrine glands is the reason why its alterations cause very noticeable variations in the appearance and symptomatology of these cases. For this reason, it is necessary to evaluate different types of gigantism.

Complementary exams

Complementary examinations of anterior lobe deficiency in adults show:

1. Moderate normochromic anemia.
2. Low blood glucose.
3. Plasma cortisol with low 17-hydroxycorticoids and 17-ketosteroids.
4. Decreased TSH and T4.
5. Decreased FSH and LH.
6. Decreased Na concentration (120 mEq/L, or less) and normal serum K.
7. Skull X-ray: they give normal images when there are no intrasellar lesions. In cases of tumors, sagittal elongation or spherical shape of the chair is observed. Extrasellar extension of the mass located within the sella turcica mass is accompanied by decreased visual acuity, optic atrophy, and homonymous bitemporal hemianopia.

Etiology

The etiology of anterior lobe deficiency in adults is varied (see Fig. 72.5).

The most common cause is chromophobe cell adenoma. In obstetric accidents, the cause is generally postpartum necrosis due to thrombosis of the pituitary vessels, as part of the generalized intravascular coagulation syndrome.

Less common causes may be non-tumoral lesions (without fibrosis) of unknown cause, specific granulomas: syphilitic and tuberculous, internal carotid aneurysms, acute infections associated with diabetes mellitus, meningitis, skull base fractures, septic thrombosis of the cavernous sinus and trauma (for example, gunshot wound to the sella turcica).

In some cases the primary defect is hypothalamic (sarcoidosis and metastatic cancer).

Since 1952, when pituitary removal began to be used as a palliative treatment for metastatic cancer and diabetic retinopathy, the hormonal and metabolic consequences have been observed.

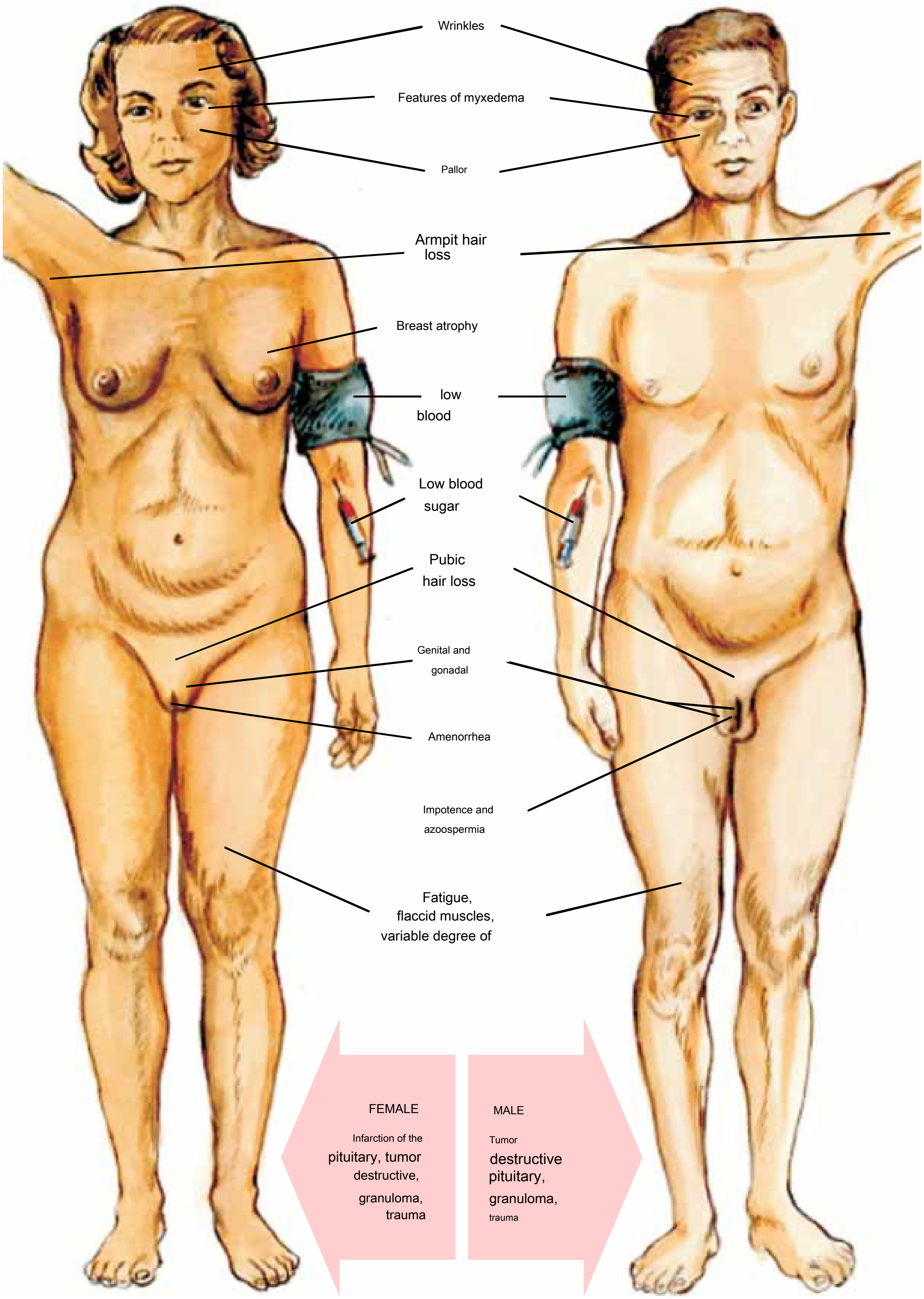

Wrinkles
Features of myxedema
Pallor
Armpit hair loss
Breast atrophy
low blood
Low blood sugar
Pubic hair loss
Genital and gonadal
Amenorrhea
Impotence and azoospermia
Fatigue, flaccid muscles, variable degree of
FEMALE
Infarction of the pituitary, tumor destructive, granuloma, trauma
MALE
Tumor destructive pituitary, granuloma, trauma

Fig. 72.5 Insufficiency of the anterior lobe of the pituitary in the adult.

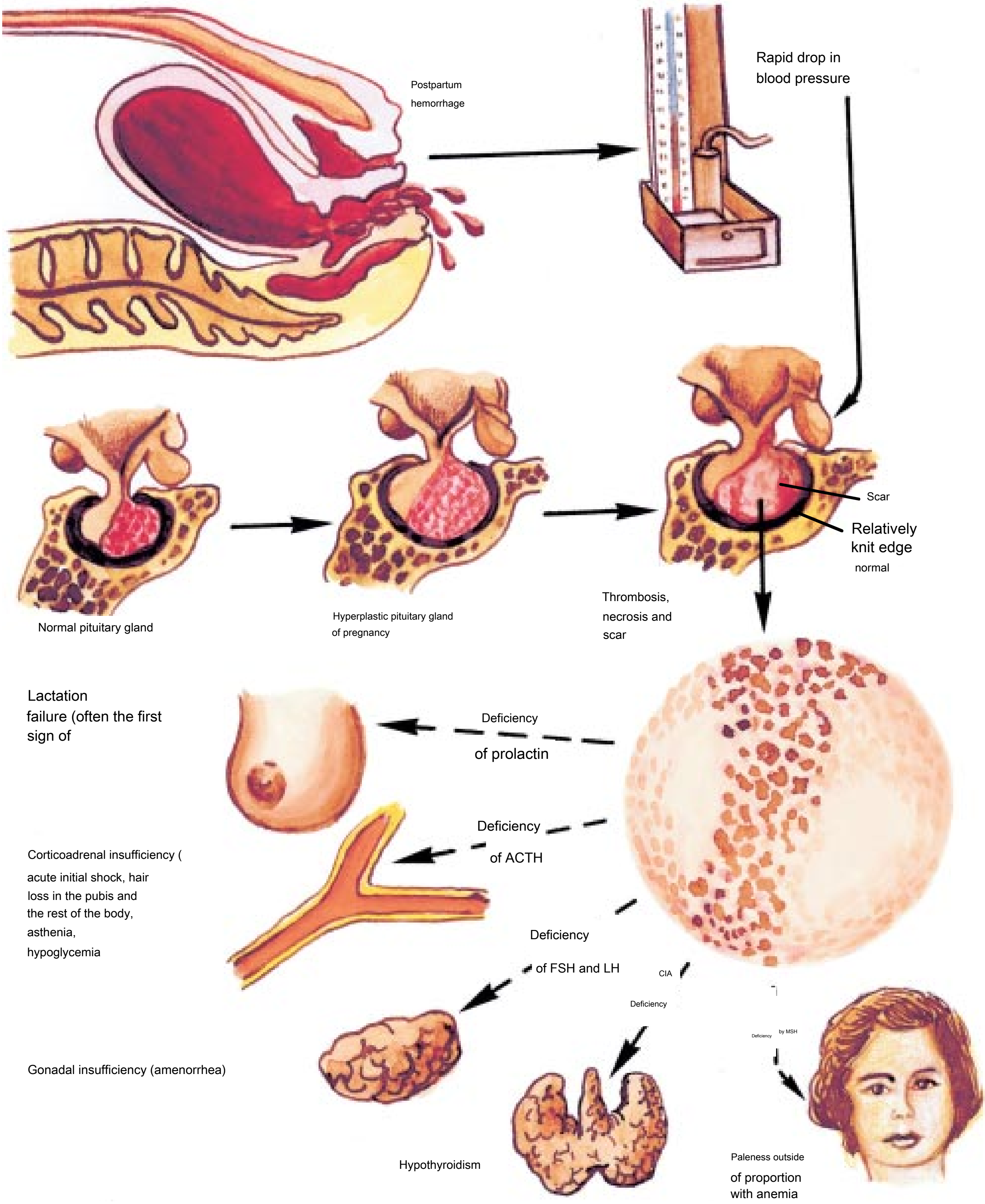

Postpartum
hemorrhage
Rapid drop in
blood pressure
Scar
Relatively
knit edge
normal
Normal pituitary gland
Hyperplastic pituitary gland
of pregnancy
Thrombosis,
necrosis and
scar
Lactation
failure (often the first
sign of
Deficiency
of prolactin
Corticoadrenal insufficiency (
acute initial shock, hair
loss in the pubis and
the rest of the body,
asthenia,
hypoglycemia
Deficiency
of ACTH
Deficiency
of FSH and LH
Gonadal insufficiency (amenorrhea)
CIA
Deficiency
Deficiency
by MSH
Hypothyroidism
Paleness outside
of proportion
with anemia

Fig. 72.6 Sheehan syndrome.

of hypophysectomy in men. It can also be seen after the application of methods to suppress pituitary function: cryosurgery, stereotaxic surgery, section of the pituitary stalk, external radiotherapy with cyclotron, etc.

Deficiency of a single anterior pituitary hormone is difficult to determine, as it may be the early phase of panhypopituitarism. While some pituitary dwarfs suffer from panhypopituitarism, others suffer from growth hormone deficiency only, of pituitary or hypothalamic origin. This fact is also observed in cases of Hand-Schüller-Christian disease, with short stature.

A prolactin deficiency occurs in women who experience insufficient lactation after childbirth.

Idiopathic gonadotropin insufficiency can manifest itself in various forms (eventually with anosmia), in males and females. Isolated deficiencies of TSH and ACTH are noted.

The common form of ACTH deficiency is observed in patients undergoing long-term treatment with corticosteroids.

Prolonged treatment with thyroid hormone and contraceptives usually induces TSH and gonadotropin deficiencies that resolve spontaneously.

Pituitary insufficiency in childhood causes dwarfism, if there is a TSH deficiency, and eunuchoidism, if the deficiency is mainly in gonadotropins.

The most common cause of hypofunction is a selective failure of gonadotropic production by the pituitary, with or without some organic lesion.

In the absence of a pituitary stimulus for testicular maturation, pubertal development does not take place and secondary sexual characteristics do not develop.

The penis remains small, with infantile proportions; The scrotum does not develop mature wrinkles and the prostate does not grow to adult size. The larynx does not increase in size and the voice maintains the high pitch of childhood.

Some pubic hair may appear, but it is usually sparse and fine, in contradiction to mature (coarse) pubic hair; nor does it extend upward along the linea alba. The axillary hair does not appear or is also scarce. Beard growth is completely absent. Frontal baldness does not develop and the hair on the head is abundant.

Because of the prolonged persistence of open epiphyseal lines, growth in height continues for a longer period than normal, particularly in the extremities, as long as somatotropic (growth) hormone secretion is adequate. The arms and legs grow disproportionately long.

Eunuchoid proportions are observed in the following measurements: the lower length of the body (from the plan- length (from the symphysis to the top of the skull), and the stroke length exceeds the standing height, although they should normally be equal (Fig. 72.7).

Osteoporosis is sometimes observed in these eunuchoid patients, presumably due to the absence of the anabolic effect on the protein possessed by testicular androgens.

They do not develop libido or potency; They are usually shy and introverted, characteristics that can often be considerably improved with appropriate hormone therapy.

In more severe cases of juvenile-onset anterior pituitary insufficiency, caused by pituitary tumors or by extrapituitary lesions (such as craniopharyngiomas) attacking the gland, the clinical picture generally includes symptoms, signs, and laboratory features of hypothyroidism and adrenal insufficiency.

These patients maintain normal body proportions thanks to low growth hormone secretion. Hypothyroidism may be an additional cause of dwarfism, either due to lack of the usual direct effect of thyroid hormone on skeletal maturation or because thyroid insufficiency leads to failure of the pituitary gland to produce adequate amounts of growth hormone. In adults, no changes occur in body proportions.

Complementary examinations of pituitary insufficiency in childhood show:

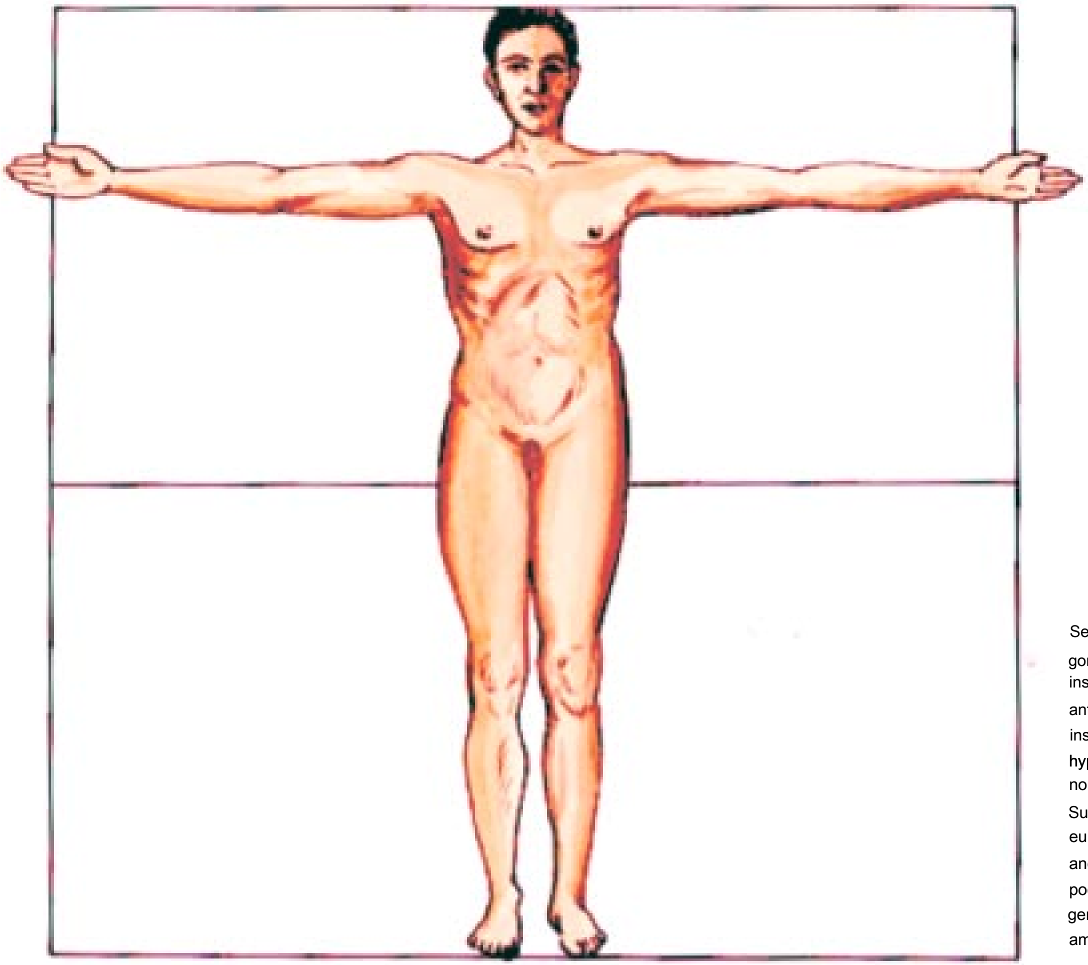
1. FSH and LH absent or decreased.
2. Decreased TSH.
3. Decreased plasma cortisol.
4. Decreased GH, no response to the insulin sensitivity test.

When dwarfism is sporadic, the deficiency may include only growth hormone or various pituitary hormones.

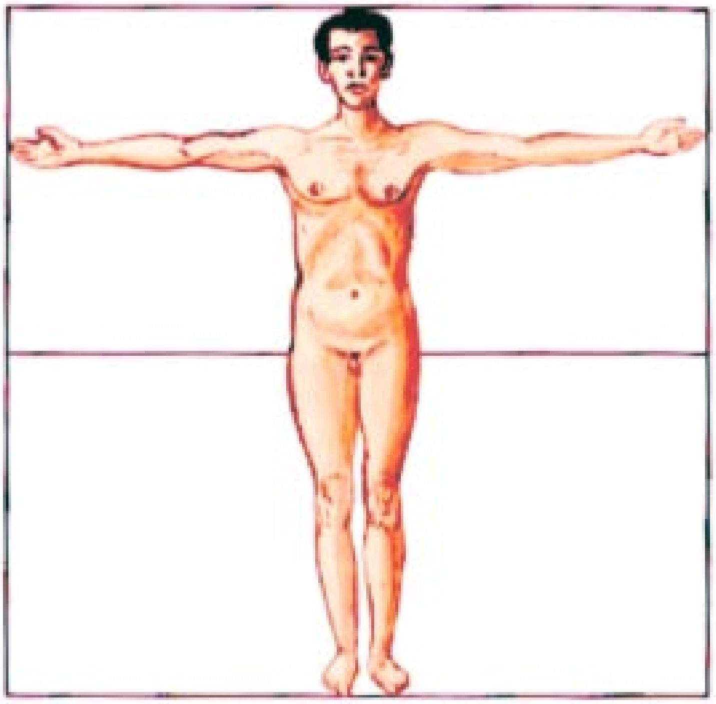
In cases of hypopituitary dwarfism, low basal plasma levels of growth hormone are observed. It is necessary to demonstrate that GH secretion does not occur in response to the hypoglycemic stimulus.

The insufficiency of growth hormone reaction to hypoglycemia is the laboratory abnormality most frequently found in cases of hypopituitarism in adults and is characteristic only of dwarfism that is associated with GH deficiency.

The etiology of pituitary insufficiency in childhood is varied. Pituitary insufficiency is a rare cause. It accounts for less than 10% of the cases. Of the tumors, the most common is craniopharyngioma. Other causes are cysts above the



Selective
gonadotropic
insufficiency (mild
anterior pituitary
insufficiency:
hypogonadism, hypogonadotropic),
normal height 0
Superior,
eunuchoid habit,
androgen deficiency,
poor gonadal and
genital development (
amenorrhea in



Insufficiency of most
anterior pituitary
hormones
including GH (
aquda anterior
pituitary insufficiency:
pituitary dwarfism),
normal body, poor
gonadal and genital
development, variable
of hypothyroidism
and adrenocortical
insufficiency, with
diabetes insipidus or without

Fig. 72.7 Anterior pituitary insufficiency in the young.

sella turcica, Hand-Schüller-Christian disease. In children who suffer from lack of maternal affection syndrome, reversible growth hormone deficiency is reported. The secretion of this hormone and gonadotropin is more easily affected than the thyrotropic or corticotropic function of the pituitary gland. Furthermore, this syndrome can be caused by the infectious, inflammatory or traumatic lesions indicated in adult hypopituitarism. Idiopathic pituitary fibrosis has been observed.

POSTERIOR LOBE SYNDROME

DIABETES INSIPIDA

Concept

It is characterized by the persistent and abundant emission (above 3 L) of abnormally clear and dilute urine, which does not contain abnormal bodies, especially sugar. It is linked to a deficiency in secretion of the pituitary antidiuretic hormone, resulting from processes that damage the neurohypophyseal system.

Syndromogenesis or pathophysiology

Physiological summary

The intimate relationship between diabetes insipidus and postpituitary disorders was established during the study of a patient with pituitary injury caused by a firearm.

Later there were those who held the idea that the lack of the posterior lobe of the pituitary gland only determines diabetes insipidus, when the anterior lobe remains intact. Subsequently, a surgical case of accidental section of the pituitary stalk was described, with the appearance of diabetes insipidus a few hours later.

Until around 1950, it was believed that the place of production of antidiuretic hormone was the posterior lobe of the pituitary gland or neurohypophysis, but a few years later, it was known that it originated in the ganglion cells of the supraoptic and paraventricular hypothalamic nuclei and that from there it is transported through the supraoptic pituitary tract to the neurohypophysis. 1a which represents the only place of storage and release of the hormone.

The specific stimulus that produces the release of this hormone is the increase in serum osmolarity. Other non-specific stimuli act through the nervous pathways, for example, pain, excessive exercise, anesthesia, nicotine, alcohol, etc.

To understand the pathophysiology of diabetes insipidus, let us remember the concepts of renal excretion (treatment). discussed in Chapter 66) and the physiological role of antidiuretic hormone (discussed in Chapter 68).

The mechanism of water excretion begins with glomerular filtration, which carries with water all plasma constituents (with the exception of proteins) in a concentration equal to that of the extracellular media. The glomerular filtration rate rises in men to 120 mL/min, that is, 180 L in 24 hours.

This considerable mass undergoes, in the course of its tubular conduction, a significant reduction of the order of 99%: a so-called obligatory reabsorption, first in the proximal tube, which is produced by an active reabsorption of electrolytes and glucose that gives rise to a passive and parallel reabsorption of water, and which is subject to the usual laws of osmosis and simply requires the integrity of the kidney cells. Perhaps it is influenced by adrenal mineralocorticoids; The action of these hormones seems to be exerted, however, with a predilection at the level of the distal tube.

The rest of the filtrate is reabsorbed in the distal convoluted tube and in the collecting tube by an active intervention that acts, not on the electrolytes, but on the water itself and which is subject to the action of the antidiuretic hormone. This results in facultative reabsorption that allows only 1,200-1,500 mL of water to reach the bladder, which constitutes the physiological urinary emission.

Antidiuretic hormone (ADH), also called pitresin or vasopressin, plays an essential role in determining the volume of urinary excretion.

As already said, its place of production is located in the nuclei of the hypothalamus.

Ablation of the posterior pituitary gland, when it spares the tuber nuclei, causes transient polyuria, but never gives rise to persistent diabetes insipidus (Camus and Roussy).

The isolated destruction of the supraoptic nuclei causes, on the contrary, around the fifteenth day, permanent diabetes

ADH also has a hypertensive action, a constricting effect on blood vessels and a potential excitomotor der on smooth muscles. This effect is different from that of oxytocin, also extracted from the posterior lobe.

LaADH exerts its action on the distal tube of the nephron. Its effect is only antipolyuric, acting on the facultative reabsorption of water and manifesting itself on the kidneys, which present polyuria with weakly concentrated urine.

The secretion of ADH normally depends on the concentration of electrolytes in the blood plasma. It is caused by the elevation of the osmotic pressure of the plasma and slowed by its descent. The injection of a hypertonic saline solution also determines a increased hormonal secretion that increases water reabsorption and dilutes the concentration of blood electrolytes.

The injection of a hypotonic liquid, on the other hand, inhibits hormonal production.

The neurohypophyseal response seems to be conditioned by stimuli coming from osmoreceptors located in the anterior hypothalamus and in the vascular territories, mainly those of the internal carotid.

Of the endocrine correlations, the thyroid plays a restricted role, at least in the physiological state. Without a doubt, thyroid hormones have a diuretic action that opposes that of ADH, but their influence is negligible in the normal subject.

Adrenocortical correlations are, however, extremely important:

1. Experimentally, the destruction of the hypothalamic nuclei does not determine diabetes insipidus, only when the anterior pituitary gland and the adrenal cortices are spared. Hypophysectomy and bilateral adrenalectomy compensate for the effects of the disappearance of ADH.

2. Analogous facts have been observed in the clinic. The development of adrenal insufficiency causes a reduction in urinary volume in diabetes insipidus. Conversely, injection of deoxycorticosterone acetate (DOCA) or cortisone sometimes increases polyuria.

3. The nature of this antagonism has been better specified. Adrenocortical steroids would act on ADH secretion through three mechanisms.

   Two of them are direct mechanisms:

   a) At the level of the nephron, reducing the sensitivity of kidney cells to ADH (Verniory).
   b) At the level of the hypothalamus, inhibiting the production of this hormone (Dingman, Despointes and Gaunt).
   c) The indirect mechanism that acts on ADH secretion is hydrosaline retention, which increases plasma volume and, therefore, reduces ADH needs.

Glucocorticoids (cortisone type) would be particularly more responsible for the direct mechanisms and mineralocorticoids (aldosterone type) for the indirect mechanism.

The notion of a hydrosaline balance must prevail, however, over that of water balance alone. It allows us to consider the problem from a fairer angle than that of a simple antagonism.

The posterior pituitary and ADH, on the one hand, and the anterior pituitary, glucocorticoids and mineralocorticoids, on the other, constitute the two groups of members of a system responsible for ensuring hydromineral homeostasis. They achieve this: the first, acting especially on the volume of aqueous reabsorption; the second, acting especially on the volume of electrolyte and more particularly salt reabsorption.

In the pathophysiology of diabetes insipidus, the mechanism of polyuria insipidus and the mechanism of symptoms must be considered successively.

Mechanism of polyuria insipidus

Pathological polyuria belongs to two types:

1. Polyuria called osmotic. They are due to a decrease in proximal and obligatory reabsorption and are characterized by an excess of partially or totally reabsorbable substances. Urine is not hypotonic.

   Its volume is not reduced by the ADH.

   Three causes dominate its etiology: sugar diabetes or mellitus, nephropathies, and certain endocrinopathies, especially adrenal or parathyroid.

2. Polyurias called insipidus (hypotonic polyurias).

   They are linked to a decrease in distal or facultative reabsorption and are characterized by the emission of hypotonic urine that does not contain normally reabsorbable substances. These polyurias are due to a lesional or functional inhibition of the ADH producing centers and constitute diabetes insipidus.

Mechanism of symptoms

1. Polyuria. It represents, with the exception of certain cases that will be specified later, the initial phenomenon. It is the consequence of insufficient aqueous reabsorption in the distal tube of the nephron.

2. Polydipsia. It is a manifestation of cellular dehydration caused by excessive polyuria. The loss of water that characterizes diabetes insipidus is not accompanied by a parallel loss of blood electrolytes and, especially, sodium. According to the laws that regulate the hydroelectrolyte balance, we know that the elevation of plasma osmolarity determines a loss of intracellular water that causes signs of dehydration of the cellular sector, especially thirst.

Syndromography or positive diagnosis

Clinical picture

Polyuria and polydipsia can be so spectacular that there are adult patients who remember the exact date and even the precise time they began.

1. Polydipsia. Dominate the scene. It is a thirst:

a) Irresistible, which forces the patient to absorb day and night an abnormally high amount of no matter what liquid: water, wine, alcohol, medicinal solution (Trousseau) and even urine. This true "thirst madness" that usually reaches 4-10 L per day, can go up to 20 or 40 L in extreme cases. She is, despite her excesses, admirably tolerated, even if it involves alcoholic liquids.

b) Invincible, does not accept any restrictions. The thirst test, which makes this character evident, constitutes, on the other hand, an extremely dangerous test. It quickly gives rise to severe signs of acute dehydration.

2. Polyuria. It constitutes the counterpart of polydipsia. It is both:

a) Considerable, 4-10 L on average; It can reach 45 L (Pidoux) and causes increased urination.

b) Constant and uniform for each patient.

c) Exaggerated by the cold and emotions.

d) Decreased by intercurrent infections and orthostatism.

e) Abnormally clear and diluted. The very weak density is between 1,002 and 1,005. The overall concentration of dissolved substances, indicated by the cryoscopic point, is very low. The concentration is also greatly reduced for each of the substances (sodium, chlorine, urea, etc.).

f) Isolated. There are no signs of kidney disease. There is no albumen, no cylinders. Renal examination tests are normal. Blood urea is rather lower than normal. The search for sugars (glucose) is equally negative.

3. Conservation of the general condition. Weight loss is observed at the beginning of the disease, but weight stabilizes quickly. Appetite is normal. The heart and blood pressure do not present pathological disorders.

Certain signs of dehydration can still be discovered in most cases, such as dry skin and mouth, constipation, decreased sweating and unstable temperature trends.

They find their biological reflection in the confirmation of plasma hypertonia with hypernatremia and a decrease in the global volume of extracellular and intracellular water.

The evolution is, apart from certain etiological forms, relatively benign. Even though it constitutes a true social illness, the condition is well tolerated, condition of organizing existence based on the needs of thirst. That is why these patients always seek to be or work in places where the water supply is assured.

Complementary exams

Laboratory tests are necessary to ensure the diagnosis in doubtful cases. These include:

1. Thirst test. It consists of eliminating drinks for 8 hours. The patient is carefully monitored, locked in his room; Urine is collected every 2 hours, blood is taken at the beginning and end of the test, which is well supported in the case of primitive polydipsia. The volume of urine decreases, the osmolarity of the last sample rises to 1,020 or even more, while the concentration of plasma electrolytes is not modified.

The test cannot, on the contrary, be prolonged more than 3-4 hours in the case of diabetes insipidus, because manifestations of acute dehydration with torturous thirst, intense agitation and hyperthermia occur. On the other hand, the diuresis continues intensely, the osmolarity of the urine remains low and does not exceed 1,010, and the plasma electrolyte concentration rises rapidly. Serious collapse accidents may occur if the test is not stopped. It is little used currently.

The response of the normal individual is less vigorous, as diuresis decreases and plasma concentration increases compared to those who suffer from diabetes insipidus.

2. Response to pitresin (ADH or exogenous vasopressin, to rule out nephrogenic causes of hormonal deficiency. Pitresin in aqueous solution is administered intravenously for one hour, drop by drop, or 5 U of long-acting pitresin tannate in oil are administered intramuscularly.

In cases of diabetes insipidus, urine output decreases by approximately 80% (Fig. 72.8).

3. Test or carbamazepine test. 600 mg of carbamazepine is administered daily for three days and if the patient has partial diabetes insipidus due to ADH deficiency, diuresis is reduced to 50% of the baseline value.

4. Miller test: It consists of the determination of plasma and urinary Osmolarity, with an increase in plasma concentration in diabetes insipidus, in relation to the normal patient.

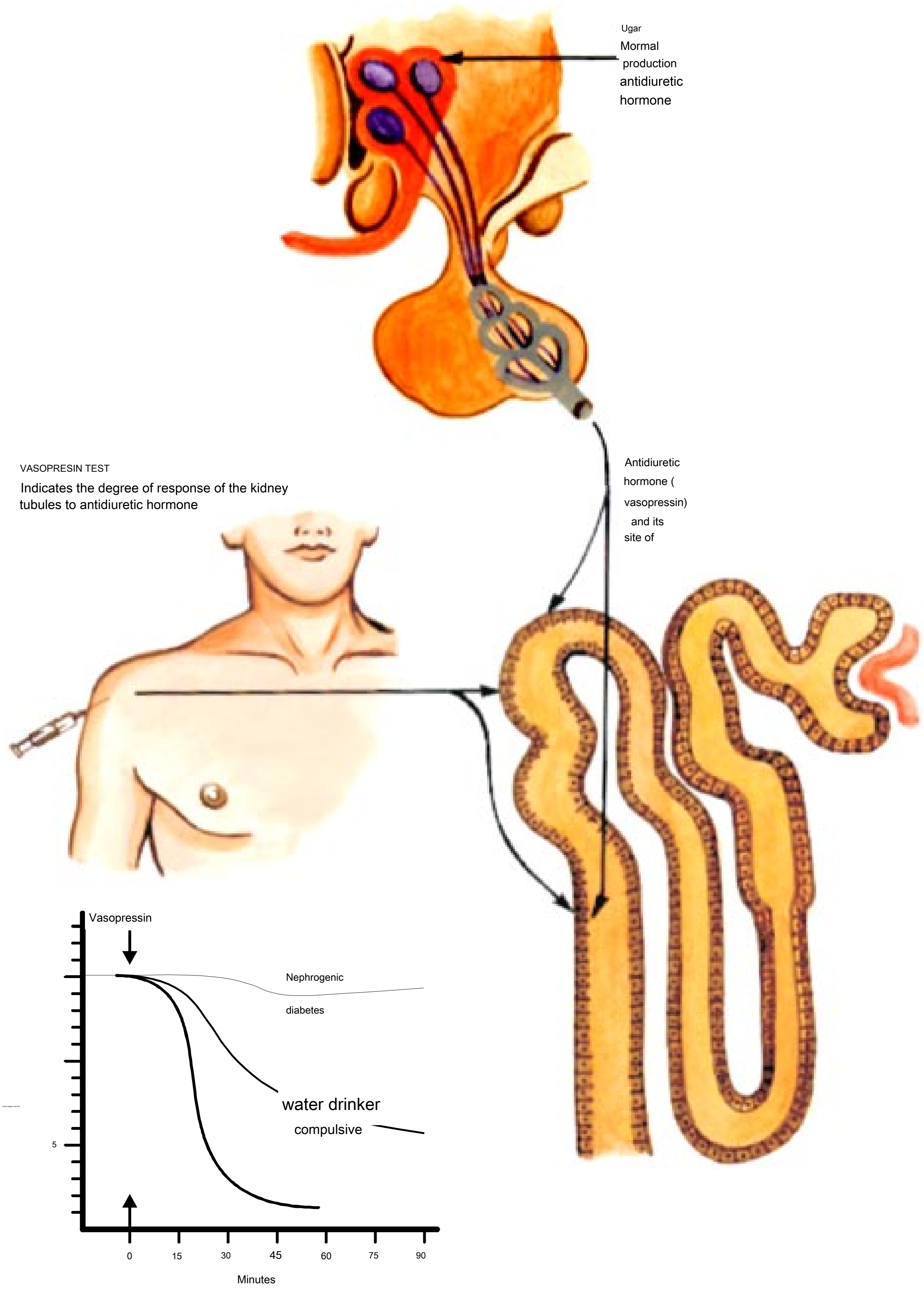

Ugar
Mormal
production
antidiuretic
hormone
VASOPRESIN TEST
Indicates the degree of response of the kidney tubules to antidiuretic hormone
Antidiuretic
hormone (
vasopressin)
and its
site of
Vasopressin
Nephrogenic
diabetes
water drinker
compulsive
5
0
15
30
45
60
75
90
Minutes

Fig. 72.8 Vasopressin test for the diagnosis of diabetes insipidus.

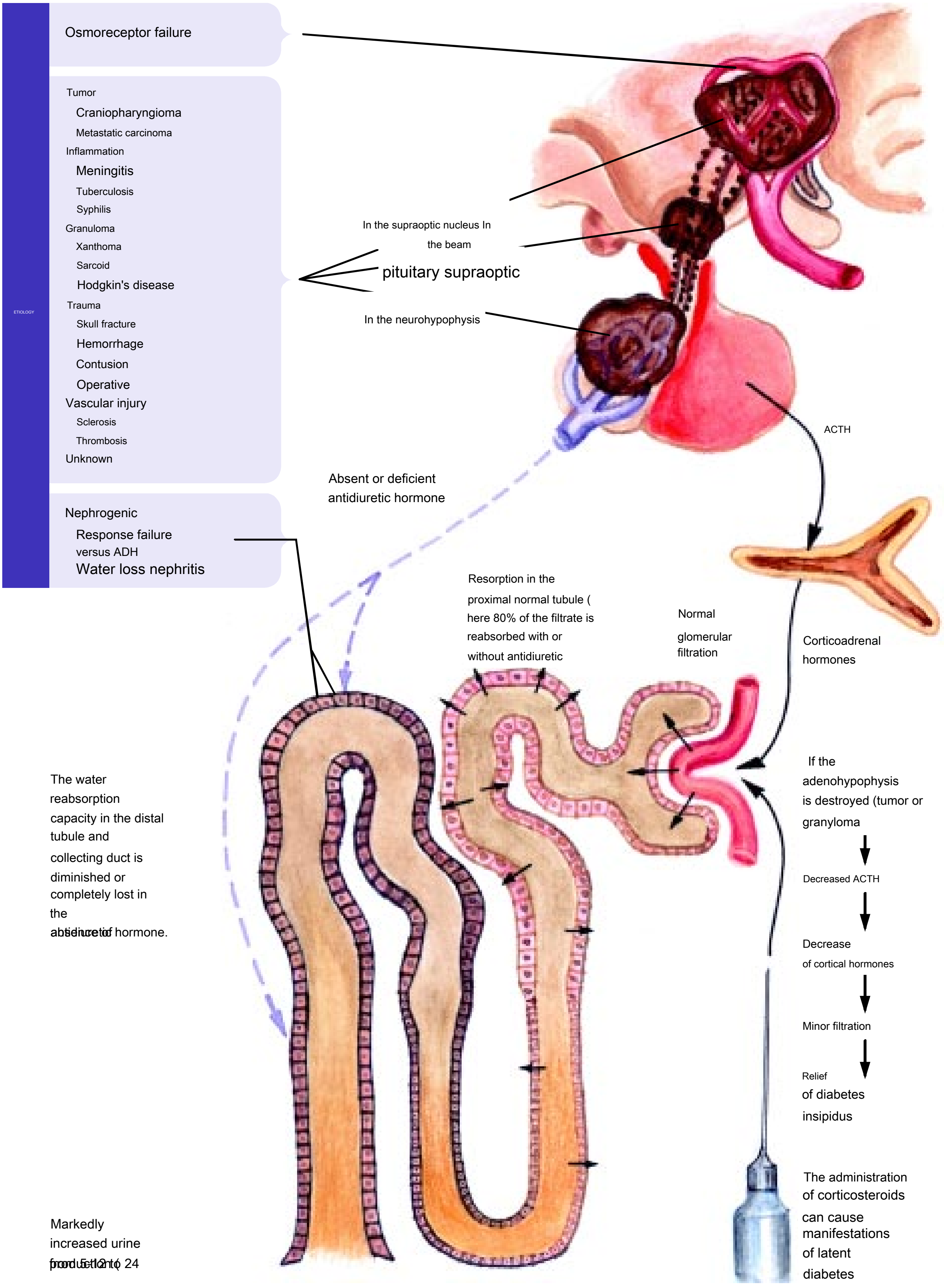

ETIOLOGY
Osmoreceptor failure
Tumor
Craniopharyngioma
Metastatic carcinoma
Inflammation
Meningitis
Tuberculosis
Syphilis
Granuloma
Xanthoma
Sarcoid
Hodgkin's disease
Trauma
Skull fracture
Hemorrhage
Contusion
Operative
Vascular injury
Sclerosis
Thrombosis
Unknown
Nephrogenic
Response failure
versus ADH
Water loss nephritis
In the supraoptic nucleus In
the beam
pituitary supraoptic
In the neurohypophysis
ACTH
Absent or deficient
antidiuretic hormone
Resorption in the
proximal normal tubule (
here 80% of the filtrate is
reabsorbed with or
without antidiuretic
Normal
glomerular
filtration
Corticoadrenal
hormones
The water
reabsorption
capacity in the distal
tubule and
collecting duct is
diminished or
completely lost in
the
absence of antidiuretic hormone.
If the
adenohypophysis
is destroyed (tumor or
granyloma
Decreased ACTH
Decrease
of cortical hormones
Minor filtration
Relief
of diabetes
insipidus
The administration
of corticosteroids
can cause
manifestations
of latent
diabetes
Markedly
increased urine
production (from 5 to 12 to 24

Fig. 72.9 Etiology of diabetes insipidus.

Etiology

It can affect both sexes and any age, although it is rare in young children; sometimes it begins in second childhood. It is usually familiar.

Various causes can cause diabetes insipidus (Fig. 72.9):

1. Trauma:
   a) Trauma to the base of the skull, due to a fall, fracture or gunshot wound.
   b) Operative trauma linked to an intervention in the region of the third ventricle.
   c) Obstetric trauma.
2. Total hypophysectomy. It also gives rise to polyuria insipidus in almost two thirds of cases.
3. Tumors:
   a) Metastatic tumors, especially breast tumors.
   b) Adenohypophyseal adenoma.
   c) Rathke's pouch tumor (craniopharyngiomas) and third ventricle tumor. Since the posterior and anterior pituitary glands have stopped blood flow, injury to the latter does not necessarily have to be accompanied by insufficiency of the former. An expansile injury should be suspected when insufficiency of both is present.
4. Infections. Encephalitis, meningitis, tuberculosis and syphilis.
5. Granulomas. Sarcoidosis, xanthomas and Hodgkin's disease.
6. Nephrogenic diabetes insipidus. It is a renal tubular defect, inherited mainly in men, in which the tubules do not respond to antidiuretic hormone.
7. Osmoreceptor insufficiency.
8. Idiopathic diabetes insipidus. Failure in the etiological search is extremely common. It is observed in two thirds of the cases. The diagnosis of idiopathic diabetes insipidus should be made after ruling out other causes and always with skepticism.

ENDOCRINOMETABOLIC SYSTEM. THYROID AND THYROID SYNDROMES

73

THYROID ANATOMY

The thyroid is an internal secretion gland that is located in the anterior region of the neck and is made up of two lateral lobes joined at their base by an isthmus.

Thyroid relationships in the neck are of great clinical value. Inside and behind it is the larynx and trachea, to which it is attached, which is why it rises and falls during swallowing. On the outside, the most important relationship is with the carotid; It is also related to the recurrent nerves.

These anatomical relationships must be taken into account since there are thyroid conditions that cause an increase in its volume and that can cause symptoms due to compression: aphonia, dyspnea (such as cancer or inflammation of the thyroid), as well as dangers of injury during surgery of this gland.

The parathyroid glands appear as two pairs of structures: one pair located in the posterior part of the upper pole of the thyroid lobes and the other pair, below the lower poles or included in their substance. This explains the frequency with which the parathyroids are affected or removed in thyroidectomies and give rise to secondary hypoparathyroidism syndromes.

Irrigation and innervation. The thyroid is such a vascularized gland that it has a blood supply five times greater than the kidney, an organ that is always used as an example of active circulation. It is supplied by the two superior thyroid arteries, branches of the external carotid: the two inferior thyroid arteries, branches of the subclavian, and a middle thyroid artery; The blood returns to the heart through the veins of the same name. The innervation is of a neurovegetative type and is in charge of the sympathetic (stimulating) and the vagus (inhibitory).

THYROID EMBRYOL0GY

At the end of the third week, a tubular invagination of the root of the tongue occurs, which in the child forms the apex of the lingual *V" and is called foramen cecum. It grows downward in front of the trachea and the thyroid cartilage until it reaches the position it occupies as an adult gland.

If the emigration of this invagination is not carried out well, the so-called accessory thyroid glands may appear, which if they subsequently suffer pathological processes can lead to clinical errors. Those capable of producing hormones are called aberrant thyroids; They may be located very far from the cervical region. They have been described in the lingual, mediastinal, etc. regions.

THYROID HISTOLOGY

The thyroid is a gland that is made up of a series of vesicles or follicles formed by epithelial cells, which contain inside a substance called thyroid colloid, which is the active hormonal substance of the gland. Each of these follicles represents the anatomofunctional unit of the thyroid (Fig. 73.1).

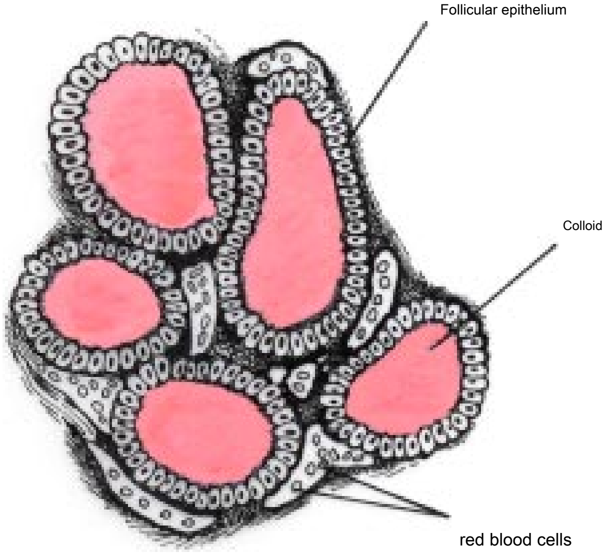


Fig. 73.1 Thyroid histology.

The thyroid is surrounded by a capsule with fibrous tracts, which penetrate inwards and are continuous with the connective tissue that exists between the follicles.

Follicle size ranges from 50-1,000 u, but is usually 300-400 u.

Each of the follicles is surrounded by a thin membrane covered by a row of epithelial-type cells whose height varies according to the stimulus that the gland receives, since they become flat and flattened when they do not receive stimulation and, on the contrary, they raise their height and become rectangular when the stimulus is intense (fig. 73.2).

Seen under the electron microscope, this epithelium shows at the apical end of the cells a large number of microvilli that are introduced into the colloid, thus expanding their contact surface. The basal end of these cells is in direct contact with the blood capillaries that surround the acinus, through a very thin basement membrane.

The cells themselves are shown during their active phase strewn with colloid vacuoles, a large nucleus, and short, thin mitochondria that vary in size and length at various phases of the cell's activity.

These mitochondria, the cell nucleus, and the Golgi apparatus move toward the apical zone during the cellular secretory phase.

Scattered within the follicles are the so-called parafollicular or "C" cells, which produce calcitonin, a polypeptide hormone that has a hypocalcemic effect.

These cells are considered to be the site of origin of a form of thyroid tumor: medullary carcinoma.

THYROID PHYSIOLOGY

THYROID HORMONES

The first problem to raise is to affirm whether one hormone or several are produced. Until 1952, thyroxine was considered the active hormone of the thyroid that exerted its physiological properties by itself.

The active ingredient is within the colloid substance, in the follicles. It constitutes the so-called thyroglobulin, which is a protein molecule of high molecular weight (around 700,000), which contains globulins of the serum type, especially gamma and alpha, and is the substrate in which thyroid hormones are contained.

Active hormones that pass into the blood are derived from thyroglobulin (through proteolysis), mainly triiodothyronine (T3) and tetraiodothyronine or thyroxine (T4).

The concentration of free T4 in the circulation is approximately three times that of T3, but the potency of the latter is three times that of T4.

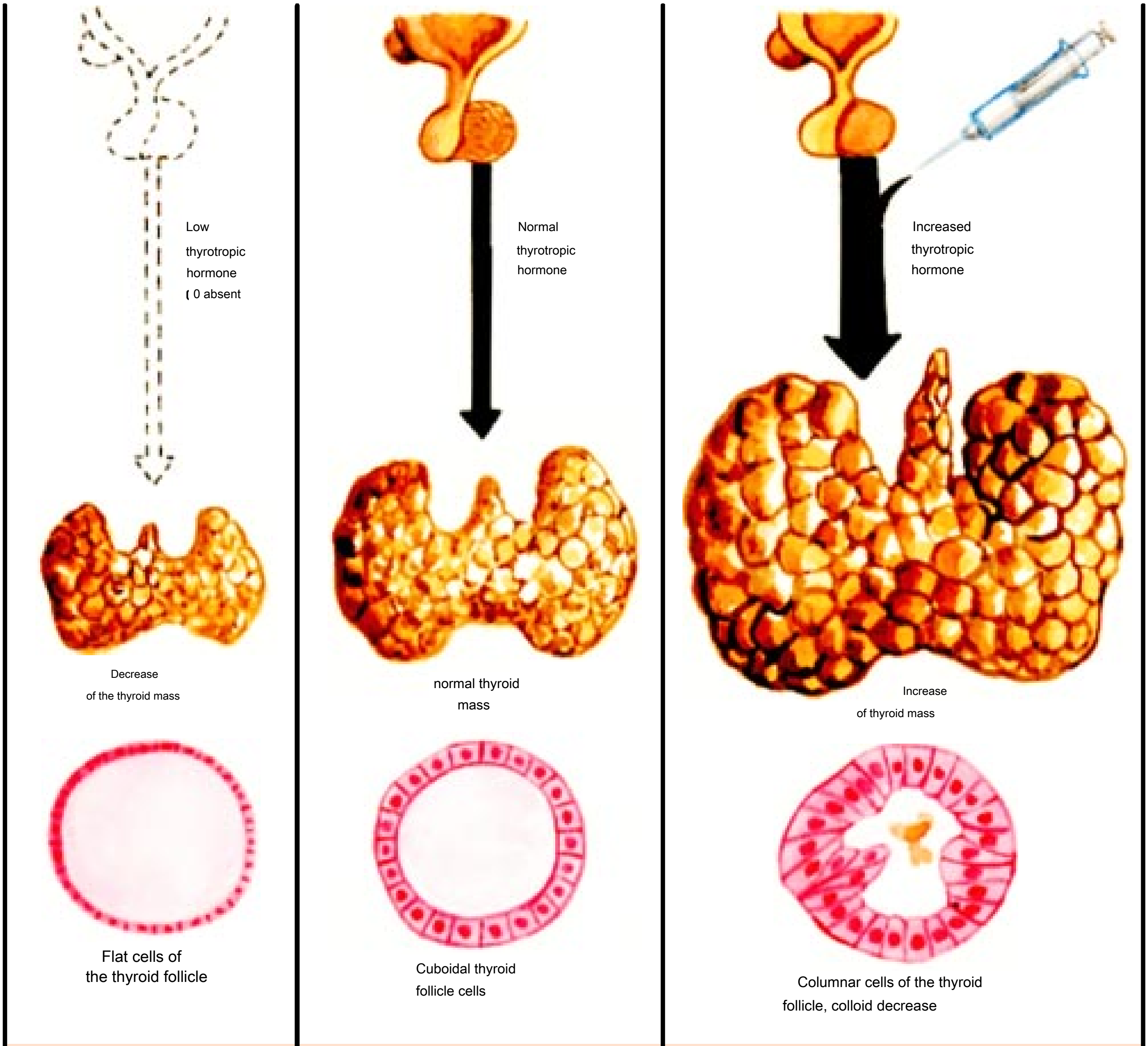
In physiological terms, both contribute equally to the metabolic function of the thyroid.

In 1957, seven iodinated amino acids had been isolated in the thyroid, of which three exist in the thyroglobulin of the thyroid colloid and four (derivatives of thyronine) pass into the blood and can be found in it. They are the following:

1. L-3 monoiodotyrosine (MIT).
2. L-3,5 diiodotyrosine (DIT).
3. L-2 or L-4 monoiodohistidine (MIH).
4. L-3, 5,3, 5' tetraiodothyronine (thyroxine or T4).
5. L-3, 5,3' triiodothyronine (T3 or triiodothyronine).
6. L-3, 3.5" triiodothyronine (RT3 or reverse triiodothyronine).
7. L-3, 3' diiodothyronine (T2).

Note: LaL means left-handed form.

Of these iodinated principles, only thyroxine and triiodothyronine have active effects.


Low
thyrotropic
hormone
(0 absent
Normal
thyrotropic
hormone
Increased
thyrotropic
hormone
Decrease
of the thyroid mass
normal thyroid
mass
Increase
of thyroid mass
Flat cells of
the thyroid follicle
Cuboidal thyroid
follicle cells
Columnar cells of the thyroid
follicle, colloid decrease

Iodine in the thyroid, normal or elevated	thyroid iodine normal	reduced iodine in the thyroid
Low GDP and EIB	Normal GDP and BEI	High GDP and BEI
T4-I3 low	Normal T4-T3	Elevated T4-T3
Capture of |low	Capture of I31 normal	Capture of 1131 elevated
Cholesterol in the blood, variable	Blood cholesterol, normal	Blood cholesterol, usually low
Null exophthalmia	Null exophthalmia	Exophthalmia almost always

Fig. 73.2 Effect of thyrotropic hormone (TSH) on the thyroid gland.

Thyroxine (T4) was considered the only thyroid hormone for many years, until 1952 when Rocht and Colas, almost at the same time as Gross and Cobbs, isolated triiodothyronine (T3), which has certain characteristics:

It acts preferentially on the gonads and on basal metabolism, in cases of euthyroid metabolic insufficiency, which are those patients with a low metabolic level and with normal thyroid function, which led us to think that thyroxine did not act well in them. In these cases, triiodothyronine acts by raising the metabolic level. Its therapeutic effects superior to thyroxine have already been confirmed. This triiodothyronine has greater diffusion power towards the tissues where it is concentrated especially in the kidney, which destroys it and transforms it into pyruvic and acetic acids and leaves the iodine free, which passes into the general circulation in the form of iodides.

Thyroid hormones are concentrated and conjugated in the liver with glucuronic acid, excreted with bile, hydrolyzed in the small intestine and reabsorbed there to enter the circulation again.

During the incubation of heart, liver and kidney sections, using labeled thyroxine, it has been possible to demonstrate how this is converted entirely to triiodothyronine, which seems to demonstrate that thyroxine is the precursor (the active hormone) of triiodothyronine.

IODINE METABOLISM

To make normal amounts of thyroid hormone, you need to ingest about 35-50 mg of iodine each year, that is, approximately 1 mg per week.

Iodine exists in the body in an amount of 50 mg, of which one fifth corresponds to the thyroid, which is the organ richest in this element. 95% of it is part of the thyroglobulin contained in the colloid substance of the thyroid follicles.

The body, even though it produces about 300 gammas of thyroxine, only needs 80 gammas of iodine daily (which are covered with ordinary food), since the rest is taken from the iodine that remains in the catabolism of thyroid hormones in the tissues and that passes to the so-called internal iodine circuit.

Iodine is ingested with water, salt and food, it is absorbed in the form of iodides by the small intestine, from which it passes into the blood and then into the extracellular fluid where it is deposited to be used as needed.

Of the iodine entered in the form of iodides, the thyroid captures the majority, approximately 90%, through an active transport mechanism capable of concentrating plasmatic iodide up to 500 times within the gland. It is the mechanism called the iodine pump. However, in a normal state this iodine pump concentrates 25 times the plasma iodine levels.

Iodine is taken up by the thyroid or salivary glands or excreted by the kidney. Uptake by the thyroid is facilitated by TSH (thyrotropic hormone) or can be blocked by perchlorate or thiocyanates.

Iodide (2I) is oxidized by an oxidizing enzyme (cytochromoxidase) and transformed into oxidized iodine (L,). This reaction can be inhibited by thiouracil and imidazole derivatives.

Oxidized iodine is rapidly utilized in the iodinization of tyrosine and forms monoiodotyrosine and diiodotyrosine. This binding can be blocked by sulfonamide derivatives.

The elements thus formed form L-thyroxine (T4) and L-triiodothyronine (T3); Both are stored in the thyroid, where they form part of thyroglobulin.

These hormones T3 and T4 are released from thyroglobulin by the action of proteolytic enzymes (activated by TSH) and are transported through the parietal cell to the blood circulation. This action is inhibited by the administration of iodine, which is used as a treatment for hyperthyroidism (Fig. 73.3). Thyroid hormones are transported in the blood by acceptor proteins (a globulin located between a., and a, and an albumin); A small part of them is in free form and is supposed to be the most active, upon reaching the cells where it exerts its effects; Finally, the hormone is metabolized in the kidney and liver.

The protein-bound hormone is known as PBI (protein-bound iodine).

This protein iodine occurs in a concentration of 3-7 gammas ()% . It is increased in hyperthyroidism, up to figures of 20 and; On the contrary, it is diminished to barely exist in cases of hypothyroidism. In the blood there is an inorganic iodine of little value.

Total iodine is 7-10% and includes all forms of iodine noted above.

Hormones reach the tissues and effector organs in the form of protein iodine, cross the cell membrane and act in the cell.

The existence of a ferment (deiodinase) capable of subtracting an iodine atom from thyroxine in the cell itself, to constitute triiodothyronine, is admitted.

Finally, the catabolism or destruction of thyroid hormones would occur, partly, in the tissues in which it has its effect and, partly, in the kidney; Thyroid hormones break down into iodides and other substances.

The iodides pass through the bile to the intestine, from this to the blood by intestinal absorption (enterohepatic circuit), from the blood to the interstitial spaces and to the thyroid, and the cycle begins again.

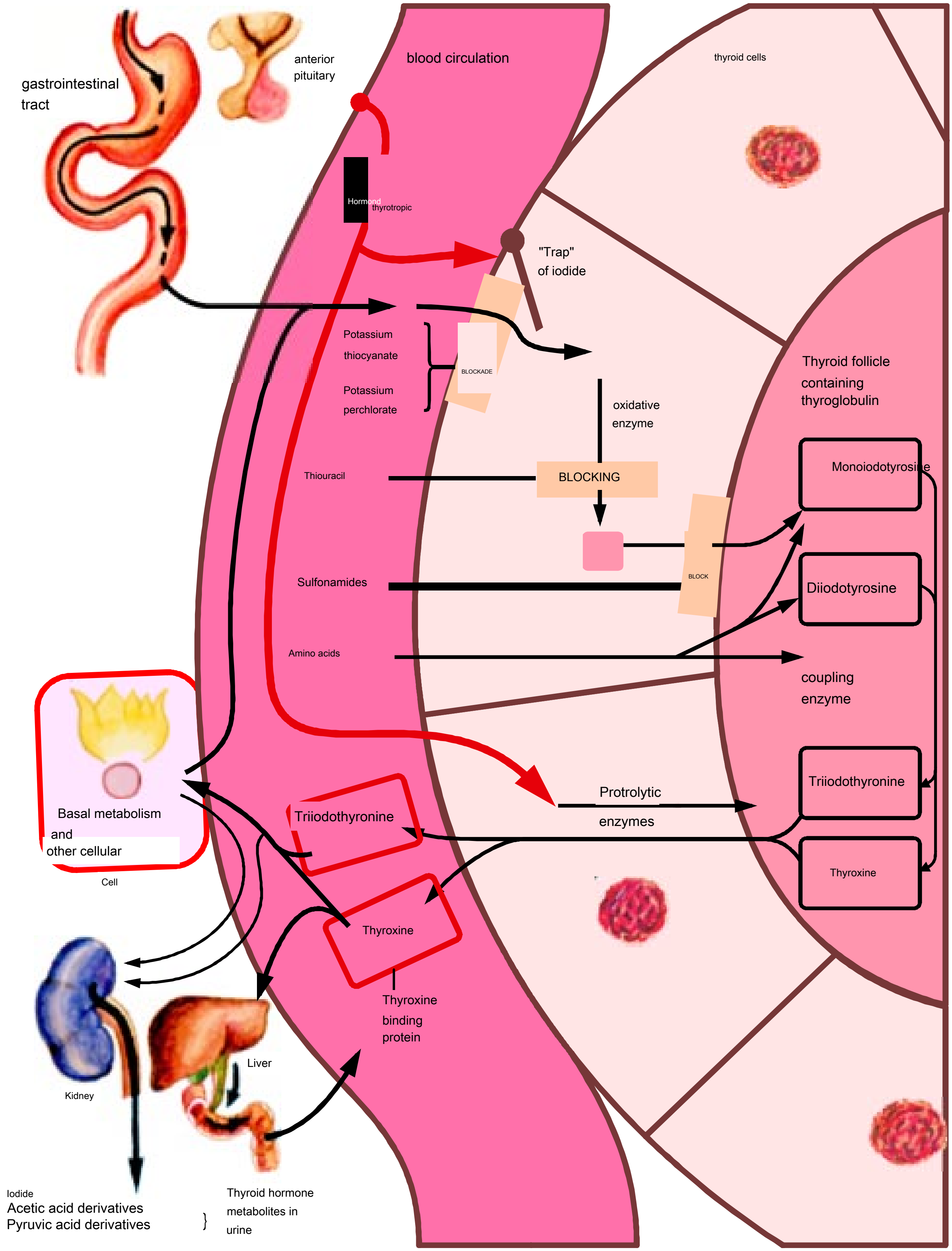

gastrointestinal tract
anterior pituitary
blood circulation
thyroid cells
Hormond
thyrotropic
"Trap" of iodide
Potassium thiocyanate
Potassium perchlorate
BLOCKADE
oxidative enzyme
Thyroid follicle containing thyroglobulin
Thiouracil
BLOCKING
Monoiodotyrosine
Sulfonamides
BLOCK
Diiodotyrosine
Amino acids
coupling enzyme
Protrolytic enzymes
Triiodothyronine
Thyroxine
Basal metabolism and other cellular
Cell
Triiodothyronine
Thyroxine
Thyroxine binding protein
Kidney
Liver
Iodide
Acetic acid derivatives
Pyruvic acid derivatives
Thyroid hormone metabolites in urine

Fig. 73.3 Biological actions of thyroid hormones.

The kidney has a great capacity to eliminate iodine. In less than 72 hours after ingestion of the radioisotope ([ ), about 80% or more is eliminated by the kidney in normal subjects, 100% in hypothyroid subjects and much less than 80% in hyperthyroid subjects.

THYROID REGULATORY SYSTEM

To maintain a normal basal metabolism, the necessary amount of thyroid hormone must be secreted constantly; For this we have a specific feedback mechanism, in which the thyroid is considered not as an isolated organ, but as part of a broad regulatory system: nervous system-hypothalamus-pituitary-thyroid-effector or tissue organ.

Let's look at the parts of this system:

1. Higher neurohypothalamic system. The central nervous system and the cortex act directly through their stimuli or through subcortical nuclei on the hypothalamus, and this intervenes as the first higher hierarchical step of the thyroid regulatory system in the control of TSH production through the releasing hormone TRH, which is found in the blood of the pituitary portal system. This factor stimulates the pituitary gland cells, increasing the secretion of TSH (Fig. 73.4).

2. Pituitary gland. It constitutes the second step in this system. The action of the pituitary gland is direct through TSH, which stimulates the uptake of iodine by the thyroid during the synthesis of thyroxine and its excretion into the blood. Thyroxine acts through a feedback mechanism, since by increasing its concentration in the blood it inhibits the production of TSH, thus maintaining balance. T3 also exerts a negative feedback effect on the hypothalamus, inhibiting the synthesis and release of TRH.

   In summary, TSH increases all known activities of thyroid gland cells. The preservation of the thyroid gland and its normal function therefore depends on the pituitary gland.

   It has been proven that when TSH is released (made by the basophilic cells of the anterior pituitary in response to thyrotropin-releasing hormone, TRH), it is transported by the blood and when it reaches the membrane of the thyroid epithelial cells, it activates adenyl cyclase and catalyzes the formation of cAMP inside the cell. Ultimately it is cAMP that stimulates all functions.

3. Other glands of the system. The adrenals and gonads also intervene. In women, estrogens inhibit the adenohypophysis, causing a decrease in TSH. In states of gonadal hyperfunction there is a decrease in TSH, while in states of hypofunction, such as climacteric, there is an increase, which may explain some climacteric hyperthyroidisms. The adrenals with their corticosteroids intervene in the retrocontrol of the hormones of the adenohypophysis.

EFFECTIVE ORGANS

They are the tissues on which thyroid hormones act, which have a greater or lesser capacity to respond to thyroxine and triiodothyronine.

THYROID HORMONES BLOCKING AGENTS

They are those agents capable of inhibiting thyroid function and which we have already studied in Chapter 71.

FUNCTIONS OF THYROID HORMONES

Thyroid physiology has been enriched by research carried out with radioisotopes, both in experimental animals and in humans, under the most varied conditions (thyroidectomy, injection of thyroid hormone or TSH). These investigations have made it possible to specify the functions of thyroid hormones and their different effects on the body. We will summarize these functions below.

Tissue oxidation function

It is the main function and the one in which the gland, through its hormones, activates the combustion of Oxygen by the cells of the body, a fact that is manifested by an increase in basal metabolism (MB).
The phrase “the thyroid is the bellows of the body” is very graphic.

metabolic functions

The action of thyroid hormones on carbohydrate metabolism is recognized: glycogenolytic effect (transforms glycogen into glucose) and hyperglycemic effect.
That is why hyperthyroidism produces a moderate state of diabetes.

It acts on protein metabolism in a catabolic sense. Regarding lipids, there are variations in the blood cholesterol level, fundamentally.
In relation to the hydrosaline metabolism, it regulates and maintains water and ions in its different compartments. Alterations in these elements may occur in thyroid disorders.

Function on the circulatory system

Thyroid hormones regulate cardiocirculatory activity by increasing heart rate, blood velocity, stroke volume, and dilating blood vessels. In a word, they activate the cardiovascular system.

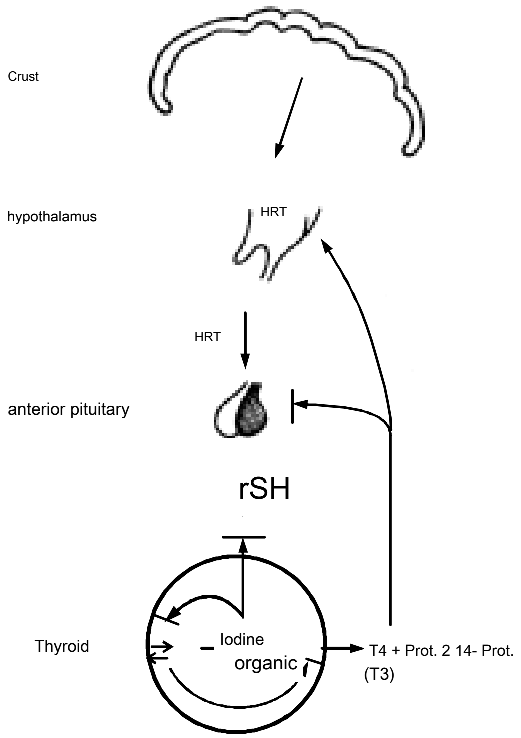


Fig. 73.4 Diagram of the factors that regulate thyroid function. In this we can see that the thyroid hormones (T3 and T4), reflected by their free concentrations in the blood, inhibit the secretion of thyrostimulating hormone (TSH) in the pituitary gland. The hypothalamic factor (TRH), which releases TSH, establishes the threshold at which this negative feedback occurs in the pituitary gland. The factors that regulate TRH secretion are not fully known, but may include influences from higher nervous centers and an inhibitory effect of thyroid hormones. Self-regulatory control of thyroid function is also shown. Elevated intrathyroid iodine concentrations decrease the rate of thyroid iodine release. Furthermore, the magnitude of the organic iodine pool exerts an inverse influence on the iodine transport mechanism and the response to TSH. That is, if the pool is high, the iodine transport mechanism and the TSH response decrease, and vice versa.

Function on the digestive system

They maintain motor and secretory neurovegetative tone. The liver plays a leading role in iodine metabolism.

Function on the renal system

Thyroid hormones do not have a specific action on this system, although in the pathological field there may be alterations.

Function on the nervous system

They maintain a state of vital tone and normal neurological activity on the cortex, subcortical nuclei and hypothalamic structures, in close correlation with the neurovegetative system.

They maintain the emotional-reactive tone of the subject and a psychological and emotional state within natural and physiological limits.

Function on the muscular system

They maintain a perfect metabolism of muscle fibers, acting indirectly on potassium metabolism during muscle contraction. Various muscular symptoms occur in thyroid diseases.

Function on the integumentary tissue

They maintain a normal state on the skin, as well as its annexes: hair, nails, etc.

Other functions

1. Action on growth and development. It has been more than demonstrated in experimental physiology work that thyroidectomy slows growth. The physiological action lies in the fact that it acts on bone growth, in close correlation with the pituitary gland.
2. Intimate correlation with the pituitary gland. It forms a system just like the pituitary-adrenal system. In both physiology and thyroid pathology, this correlation must always be kept in mind.
3. Intimate correlation with the gonads. The same criterion exists, even from the physiological point of view. States of greater thyroid excitement are found in all people: at certain moments of sexual life, such as puberty and the climacteric. In addition, thyroid hormones maintain libido, ovulatory potency, lactation, etc.

4. In blood regulation. Thyroid hormones are essential for normal erythropoiesis and spinal cord activity.

5. Other correlations. These include: age, body constitution, endocrine formula, diet and water and its richness in iodine, vitamins (especially A), iron, etc. Likewise, environmental factors influence like no other gland. Emotional psychic factors can vary the normal rhythm of thyroid hormones.

THYROID SYNDROMES

Among these syndromes we have:

Hyperthyroid syndrome (hyperthyroidism).
Hypothyroid syndrome.
-Juvenile hypothyroidism.
- Congenital hypothyroidism (thyreosis).

HYPERTHYROID SYNDROME (HYPERTHYROIDISM)

Concept

Hyperthyroidism is the name adopted for the disease whose main pathophysiological basis is the hyperfunction of the thyroid and, therefore, of thyroid hormones, which gives rise to a set of symptoms and signs, according to the reaction of the organs or tissues to said hormones.

Synonymy

Toxic goiter, exophthalmic goiter, hyperfunctioning thyroid, hyperthyroidism, Basedow disease, Graves-Basedow disease. The general name includes toxic adenoma or Plummer's disease, which despite its own characteristics, can be studied together.

Syndromogenesis or pathophysiology

This syndrome is characterized by an excess production of thyroid hormones that determines a series of symptoms and signs, secondary to exaggerated tissue metabolism.

Diffuse toxic goiter or Graves-Basedow disease, the most common hyperthyroidism, is an autoimmune disorder, characterized by the elevation in the blood of IgG immunoglobulins, called thyroid-stimulating immunoglobulins (TSI), which act on the TSH receptors in the thyroid cell and produce competitive inhibition of this hormone, which causes hyperfunction of the thyroid gland.

In turn, the symptoms can be grouped into:

1. Vegetatives. Anxiety, nervousness, tremor, psychological irritability, excessive sweating, diarrhea, dysthermia, heat intolerance, visceral vegetative disorders (circulatory, digestive, etc.).
2. Metabolic syndrome. It is due to the thyroid hormones themselves. Due to its action on organic combustion, cellular oxidation and effects on protein, lipid, carbohydrate, hydromineral and iodine metabolism, alterations are triggered that we will point out later.

Syndromography or positive diagnosis

Clinical picture (fig. 73.5)

The constituted syndrome is very characteristic. The general inspection of the patient sometimes shows his distressing appearance, with bright eyes and exophthalmia; nervousness associated with thinness, which seems strange, since the patient eats more than before. Other times it is the increase size of the thyroid gland (goiter) associated with other symptoms, or it may be diarrhea, palpitations or any other symptom that attracts attention.

It is common for several of these symptoms to be associated in an apparently healthy individual without connection to any internal or external event, which is common, or after some emotional shock, any illness, weight loss cure, marriage, pregnancy, childbirth, breastfeeding or menopause.

In hyperthyroidism with nodular goiter or toxic adenoma (Plummer's disease), the onset is more gradual and has the characteristic of manifesting itself in a person who already had a thyroid adenoma. Small nodules may go unnoticed until the disease begins.

General symptoms

On examination, an abnormal sensitivity to heat and greater tolerance to cold is found.

Weight loss is one of the cardinal symptoms, although not mandatory. This thinness is due to the increase in combustion and metabolism, which requires a greater number of calories.

The typical expression is anxiety, anguish or fear, a bright look.

The typical patient is dominated by restlessness, hyperkinesia. Even sitting, the hyperthyroid person makes frequent changes in posture (hands, arms, legs).

Thyroid signs

Enlargement of the thyroid (goiter). Little or very accentuated, it is part of the picture in most cases (figs. 73.6 and 73.7). Let's start from the fact that a 20-30 mg thyroid is normally neither visible nor palpable. Enlargement has already been studied in Chapter 71 (see "Thyroid enlargement").

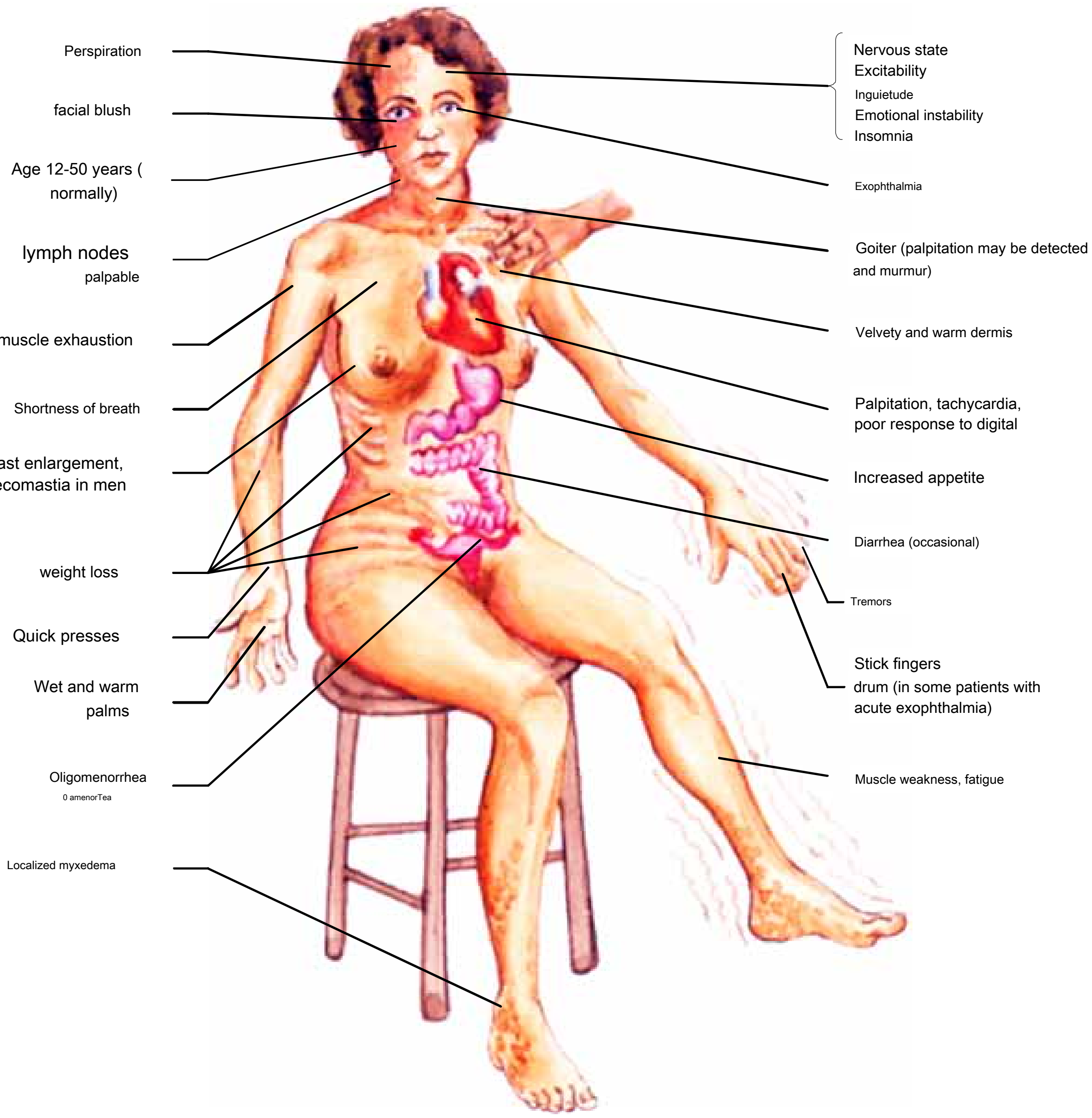
Exophthalmnia and ocular signs

They constitute cardinal symptoms, mainly exophthalmia (protrusion, procidence or propulsion of the eyeballs) associated with bright gaze. It actually constitutes the " business card" of these patients that stands out upon simple inspection (see also Chapter 71).

It is usually bilateral, although not always symmetrical, sometimes it is unilateral. It is more common in the form of diffuse goiter; may be missing in the adenomatous.

It may be the first manifestation, sometimes parallel to other symptoms, or appear late.

There is no parallel between the intensity of hyperthyroidism and exophthalmia.


Perspiration
facial blush
Age 12-50 years (normally)
lymph nodes palpable
muscle exhaustion
Shortness of breath
Breast enlargement, gynecomastia in men
weight loss
Quick presses
Wet and warm palms
Oligomenorrhea
0 amenorTea
Localized myxedema
Nervous state
Excitability
Inguietude
Emotional instability
Insomnia
Exophthalmia
Goiter (palpitation may be detected and murmur)
Velvety and warm dermis
Palpitation, tachycardia, poor response to digital
Increased appetite
Diarrhea (occasional)
Tremors
Stick fingers drum (in some patients with acute exophthalmia)
Muscle weakness, fatigue

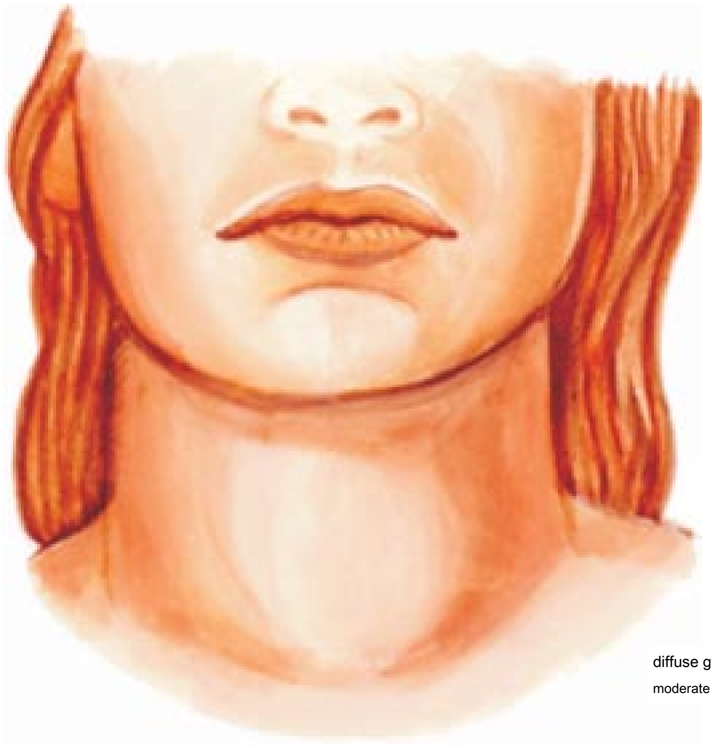
The following combinations are observed:

1. Hyperthyroidism with marked exophthalmia.
2. Hyperthyroidism without exophthalmia (common in adenoma).
3. Exophthalmia without hyperthyroidism (treated cases).

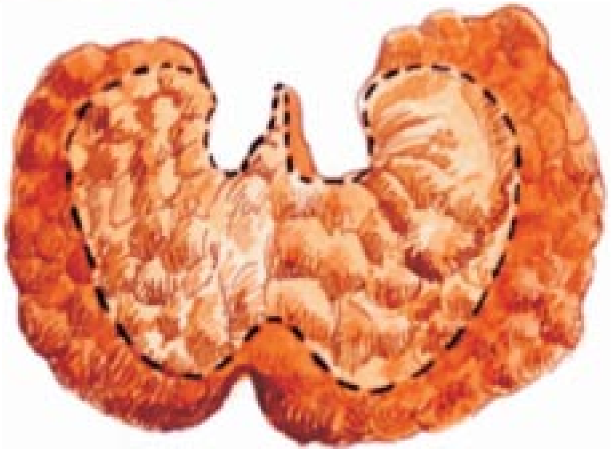
Added to exophthalmia is a functional deficit of the extrinsic muscles of the eye that affects ocular motility.

Sometimes the disease dominates the ocular picture with severe progressive exophthalmia, with intense chemosis, edema and eyelid congestion, limitation of eye movements, diplopia, corneal ulcerations that can lead to blindness, which constitutes malignant exophthalmia, hyperophthalmic hyperthyroidism ophthalmoplegia (fig. exophthalmic 73.8).

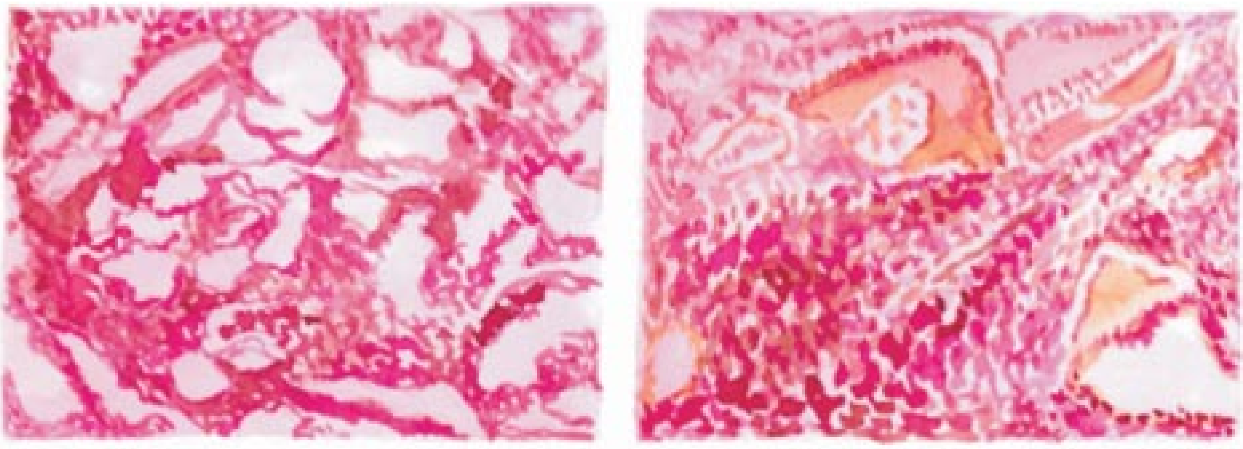
The pathophysiology of exophthalmia, especially the malignant or secondary form, is conditioned by an increase in TSH, since it produces an increase in the retro-orbital tissues (adipose infiltration, mucopolysaccharide infiltration, lymphocytic reaction) that not only affects the connective tissue of the orbit, but also the extrinsic muscles of the eye.



diffuse goiter
moderate in size



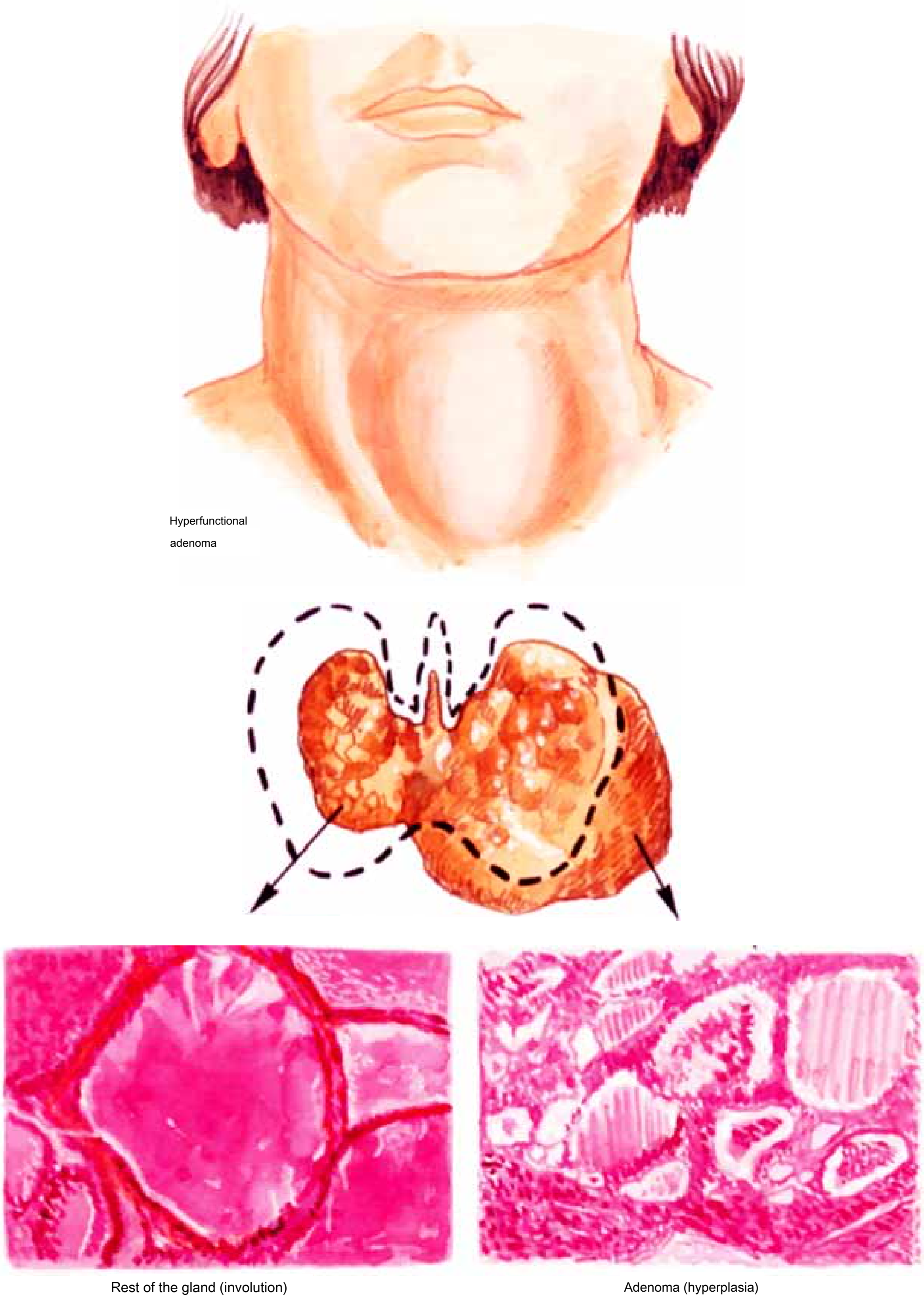
Diffuse enlargement
and congestion of the thyroid
gland (the dashed line
indicates the normal size
of the gland)



diffuse hyperplasia

Hyperplasia with lymphocytic infiltration

Fig. 73.6 Goiter and morphohistological alterations in hyperthyroidism.


Hyperfunctional
adenoma

Rest of the gland (involution)

Adenoma (hyperplasia)

Fig. 73.7 Enlarged thyroid in hyperfunctional thyroid adenoma.

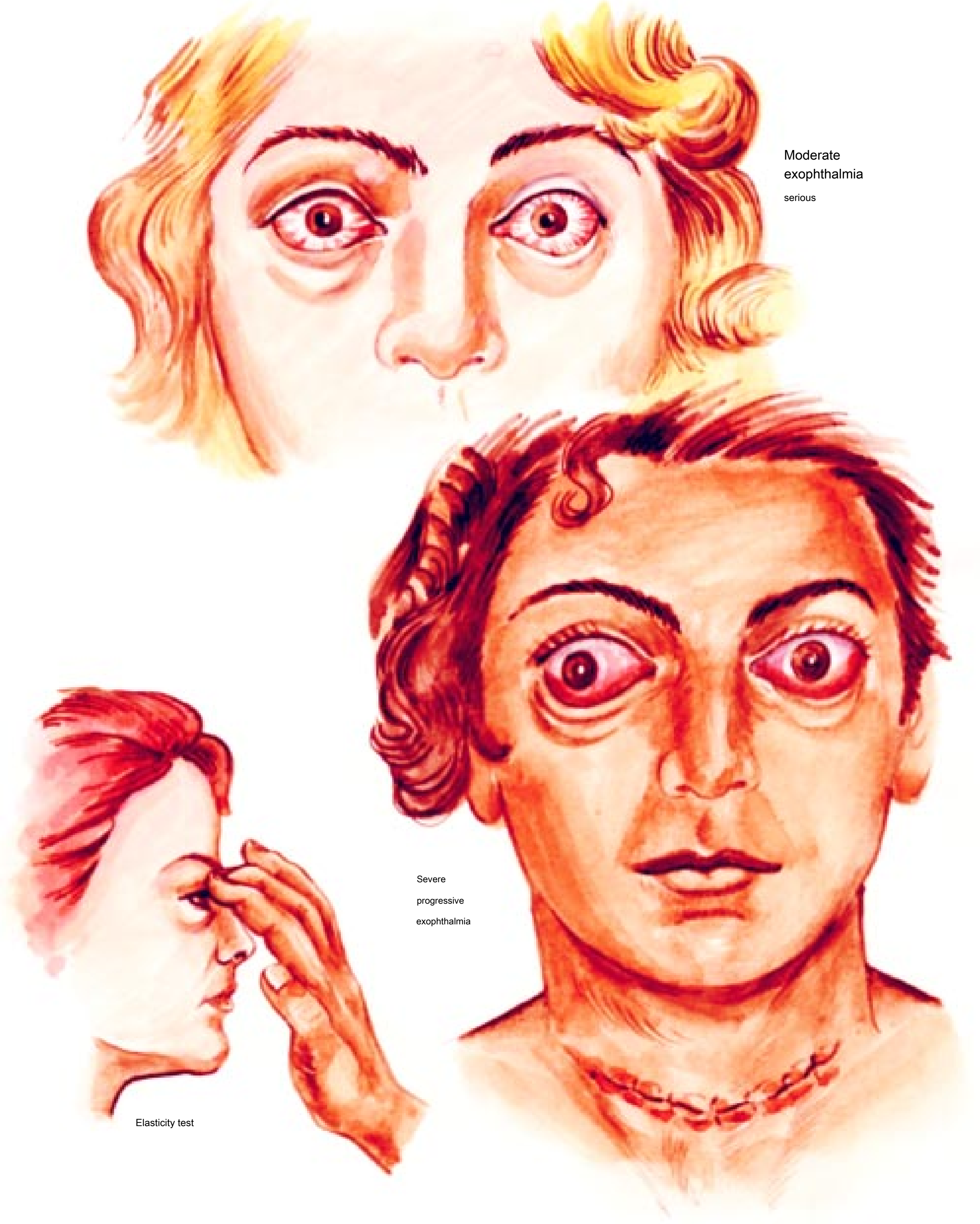

Moderate
exophthalmia
serious
Severe
progressive
exophthalmia
Elasticity test

Fig. 73.8 Degrees of exophthalmia in hyperthyroidism.

The ocular signs are explained by retraction of the upper eyelid, weakness of the extrinsic muscles, or orbital edema. THE most notable are:

1. Dalrymple sign. Increased palpebral fissure.
2. Von Graefe sign. When we direct our gaze downwards, the upper eyelid does not follow the eyeball and an exaggerated part of the sclera is exposed.
3. Stellwag sign. Reduction or abolition of blinking.
4. Möebius sign. Difficulty in convergence. When approaching an object, the eyeballs must converge; but when they reach the vicinity of the eye, instead of converging (as if moved by a spring) they head outwards.
5. Ballet sign. Complete loss of all extrinsic eye movements.
6. Rosenbach's sign. Fibrillar tremor of the eyelid.
7. Claude-Bernard-Horner sign. Eyelid ptosis, miosis, enophthalmia, produced by compressive paralysis of the cervical sympathetic nerve, due to goiter.

Cardiovascular symptoms

They almost always exist; They are derived from greater cardiac activity and greater vascular lability, due to increased combustion and greater oxygen needs.

Among them we have: palpitations, sometimes very annoying; sensation of dyspnea and precordial tightness, sometimes of anginal type.

On examination we found: tachycardia, which is one of the capital and constant findings in hyperthyroidism; It is determined by the increase in blood velocity that also sometimes causes functional murmurs.

Blood pressure can be normal or high, the minimum is usually low, with differential width that conditions some signs of aortic insufficiency (arterial dance in the neck, Musset sign, capillary pulse, etc.).

Cardiovascular tachycardia and erethism, especially in elderly people, with prolonged hyperthyroidism or with other additional heart disease, represents a marked cardiac impact that can lead to heart failure of different degrees. When these cardiovascular signs stand out over other hyperthyroid symptoms, we have the so-called hyperthyroid heart disease or cardiothyrotoxicosis.

Nervous system disorders

They are one of the most important and typical.

Tremor is one of the constant signs. When it is moderate, it affects only the hands and is evident by extending them with the fingers separated and placing a sheet of paper on the back of them.

There is psychic and emotional hyperexcitability; The sick speak lively, they become irritated for any reason. Ideation is accelerated. Sometimes there is euphoria and changes in behavior. Insomnia is common. Other times there are symptoms of depression, anguish, apathy. Sometimes there is a suicidal tendency. These alterations can lead to true states of psychosis.

There may be other nervous symptoms. Hyperactivity can be both sympathetic and parasympathetic.

The first causes cardiac hyperactivity and mydriasis; the second, hyperhidrosis and intestinal hyperperistalsis. The sensitivity to adrenaline is exaggerated; The 0.5 mg injection (Goetsch test) causes accentuation of tachycardia, paleness, increased blood pressure and a feeling of anxiety. The osteotendinous and cutaneous reflexes are lively. Cranial nerve palsies may occur.

Alterations of the loconotor system

Due to alterations in the metabolism of calcium and phosphorus, with increased urinary elimination of the same, bone alterations frequently occur.

Osteoporosis first affects the small flat bones, then the vertebrae, and then the rest of the skeleton. These alterations are due to the physiological fact of the hormonal stimulus on bone growth, sometimes very noticeable.

Muscular alterations consist of a decrease in the segmental strength of the muscle with a feeling of easy fatigue and asthenia, which is clinically evident by different maneuvers. The normal individual, sitting in a chair, keeps his leg extended for a minute, the hyperthyroid person does not hold it for more than half a minute (thyroid leg sign); Furthermore, if he is lying down, he cannot sit with his arms crossed; It is also impossible for him to stand up on a chair without holding on. Sometimes muscle atrophies and true myopathies (hyperthyroid myopathies) occur.

Digestive system disorders

Increased appetite along with malnutrition is a common finding, although anorexia may occur; sometimes there is sialorrhea; There is usually jejunoileal hypermotility, associated with diarrhea or correction of previous constipation; There may be disturbance of intestinal absorption, especially of B complex vitamins. Diarrhea may be severe in severe cases.

The liver is frequently affected although the disorders are subclinical. The lesion is located in the cell and can be due to the isolated or combined action of the depletion of the glycogen load or the lack of proteins. The symptoms are not specific. Liver tests may be positive.

Sexual disorders

In men, there is sometimes an increase in libido and sexual activity. In intense cases, libido is reduced or disappears, and there may be impotence.

In women, oligomenorrhea or polymenorrhea may occur; In intense cases, ameno- Irea is reached with lack of ovulation. Pregnancy, childbirth and breastfeeding can have a normal course, but the risks are greater. In intense cases there is sterility or early habitual abortion.

Skin disorders

The skin undergoes characteristic alterations. It is always moist and seborrheic. The skin temperature is increased. Sweating is exaggerated and is easily triggered by slight increases in environmental temperature, to which hyperthyroid patients are so sensitive.

The hair is thin and often falls out early, which is why baldness is early and common. Decreased axillary and pubic hair may also be observed.

Nails grow quickly; They are thin and fragile, with trophic alterations.

Complementary exams

1. Elevated T4.
2. Elevated T3.
3. Decreased TSH.
4. High intake of [13l, especially fractionated (at 2, 4, 6 and 24 hours).
5. Thyroid scintigraphy, especially in nodular goiters and solitary hyperfunctional nodules (Fig. 73.9).
6. Ultrasound or thyroid ultrasound: with the same indications as the previous ones.
7. Plasma determination of TSI (in specialized centers), which are elevated.
8. Circulatory examinations: minute volume, velocity and blood volume are increased.
9. In the telecardiogram there may be an increase in the cardiac area, with an increase in the left middle arch.
10. The electrocardiogram can show small non-specific disorders: large P wave in DII and DIII, elongated PR interval, high T in DI and DII and heart rhythm alterations such as sinus tachycardia and sometimes, atrial flutter or fibrillation; There may be extrasystoles, more common ventricular ones, and complete AV blocks.

Etiology

Goiter is usually caused by:

1. A diffuse primary hyperplasia of the thyroid (classic disease).
2. Hyperfunctioning focus within an adenomatous colloid goiter.
3. Benign or malignant tumor with the capacity to produce rar thyroid hormone.
4. Hashimoto's thyroiditis.

2 and 3 are less common causes. Hashimoto's thyroiditis is a rare cause.

HYPOTHYROID SYNDROME

Concept

It is a set of symptoms and signs that occur due to a deficiency in the endocrine function of the thyroid, with the consequent insufficiency of thyroid hormones.

We generally speak of hypothyroidism (although in many books it is designated by the name of myxedema), when it is an adult form, and cretinism, when it is a congenital form; the latter, with its two varieties: the endemic and the sporadic.

In reality, there is only one hypothyroid syndrome, but with different characteristics, depending on whether the condition presents in adulthood, in a growing child (juvenile myxedema) or congenitally.

Syndromogenesis or pathophysiology

The deficiency of the thyroid hormones, thyroxine (T4) and triiodothyronine (T3) acts by causing alterations of greater or lesser magnitude in almost all tissues, which depend on the intensity of said deficiency. In order not to fall into repetitions, we refer the reader to the physiology and action of these hormones indicated at the beginning of this chapter.

Syndromography or positive diagnosis

Clinical picture (fig. 73.10)

It is very varied; As in hyperthyroidism, the clinical picture may present complete or forms in which some of the symptoms that will be presented are missing.

mos.

There are also the poorest forms in their clinical richness, called monosymptomatic or oligosymptomatic, which are of great interest in daily practice, since they can go unnoticed.

The general inspection is of great value and sometimes with it we need a diagnosis, since it is a person with a tendency to obesity, pale, with coarse features, with special apathy and indifference.

Facies (fig. 73.11)

Typical, rounded in appearance, with bloated eyelids, with increased new folds on the face

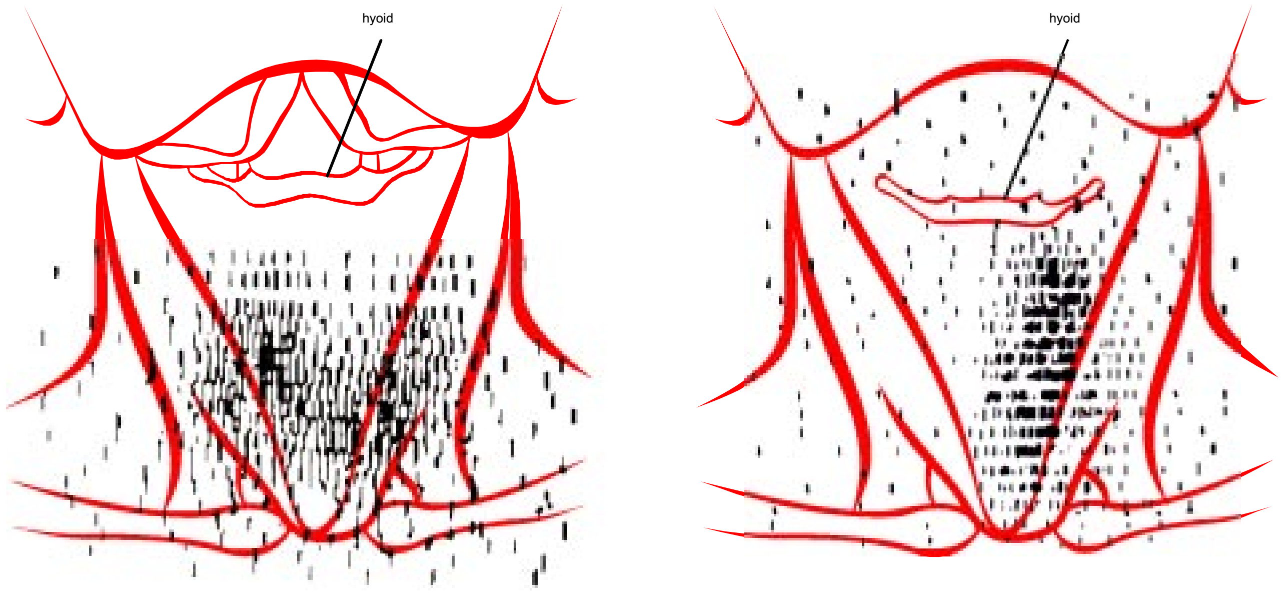


Fig. 73.9 Thyroid scan: a, in diffuse hyperthyroid goiter; b, in hyperfunctional thyroid adenoma.

and disappearance of the normal ones; The face is expressionless, apathetic, yellowish in color, with little hair, especially the tails of the eyebrows; The tongue is large, sometimes with dental impressions on its edges. Thick lips, especially the lower one, which is somewhat drooping. Poorly preserved teeth, frequent cavities.

Integumentary tissue

The skin is typical and characteristic. It impresses with its pale, yellowish color; It is thick and coarse and on palpation it is cold, dry, rough and hard (hyperkeratosis);
Pruritus, pyodermitis, scleroderma, vitiligo, etc. can occur on this skin (see Fig. 73.11).

The hair becomes dry, fragile, brittle;
there is early baldness.

The nails appear striated, rough, with atrophic signs. The role that hypovitaminosis A may have in this skin syndrome is pointed out.

Myxedema

We understand by myxedema the mucous infiltration of the tissues that gives the sensation of edema, but is harder than this and generally leaves no trace (godet), when pressed on a resistant plane. Its biochemical structure is that of a mucilaginous or mucoid substance; hence the name myxedema.

In reality, it is a mucilaginous invasion of the interstitial tissue by a mucopolysaccharide, chemically similar to that of connective tissue.

Three chemical compounds are found in this mucopolysaccharide:

1. Mucin (high molecular weight protein molecule).
2. Chondroitin.
3. Hyaluronic acid.

Mucopolysaccharides bind water and cause myxedema, which can be generalized to the entire body in the typical forms of hypothyroidism, but is especially observed on the face and ankles. It can be found in: heart, larynx, kidney, liver, nervous system and serous glands. In this last case it determines states of polyserositis and true anasarcas.

It is the myxedema that gives the patient that flabby and obese appearance; In reality, it is false obesity, since after specific treatment it can take on an appearance of normal nutrition and even thinness.

They are apathetic, indifferent patients, who have great tolerance to heat and great sensitivity to cold (subjectively and objectively), since the tendency to hypothermia is proven, thermometrically.

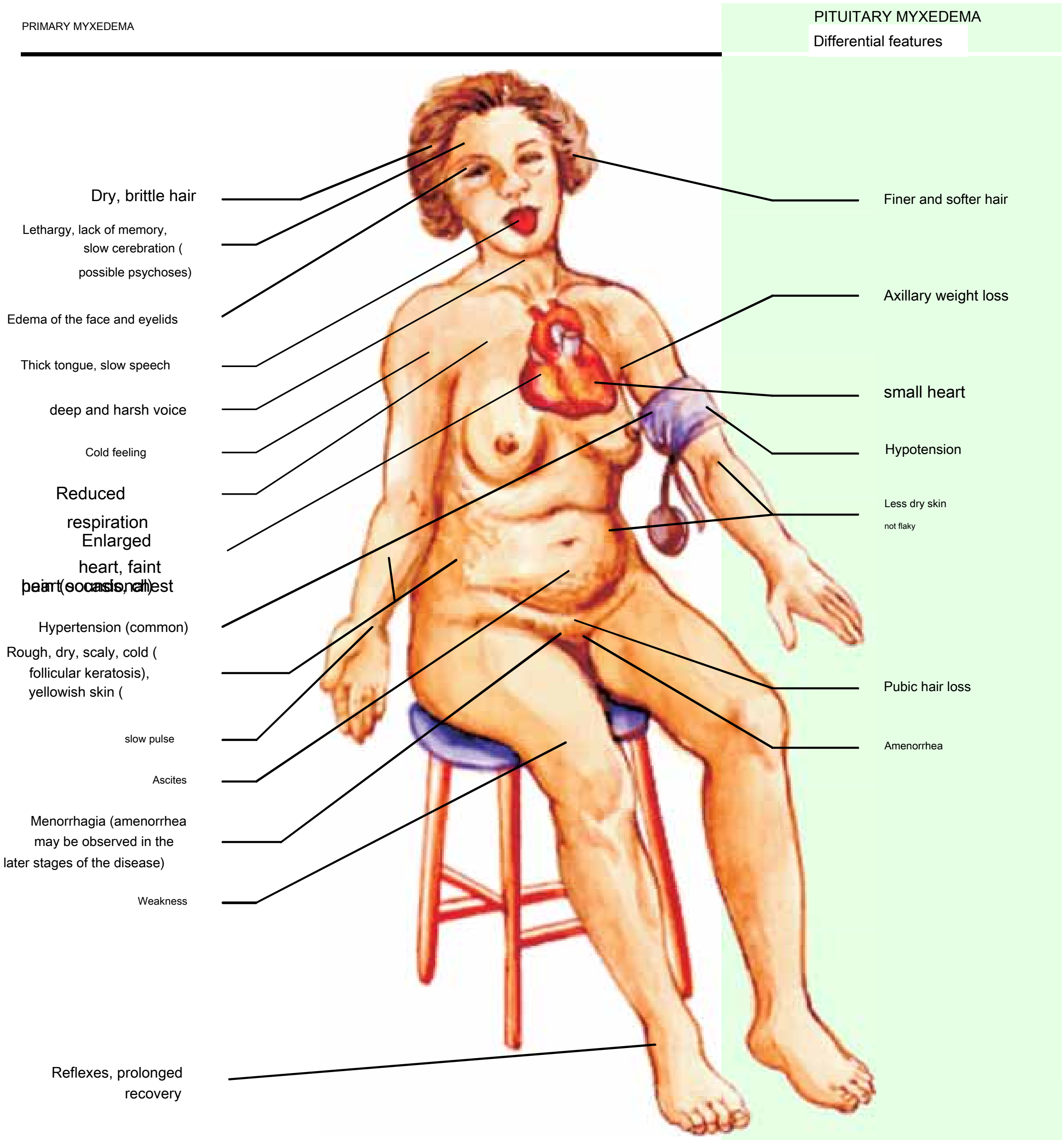
The thick, monotonous, tired, poorly modulated voice is due to laryngeal infiltration.

The coarse appearance, with thick and widened hands and feet, short and wide fingers and a special palmoplantar keratoderma, is reminiscent of acromegalics.

Patients do not complain about anything, perhaps because of their apathy. Sometimes they report muscle fatigue, physical and mental asthenia, and great laziness.

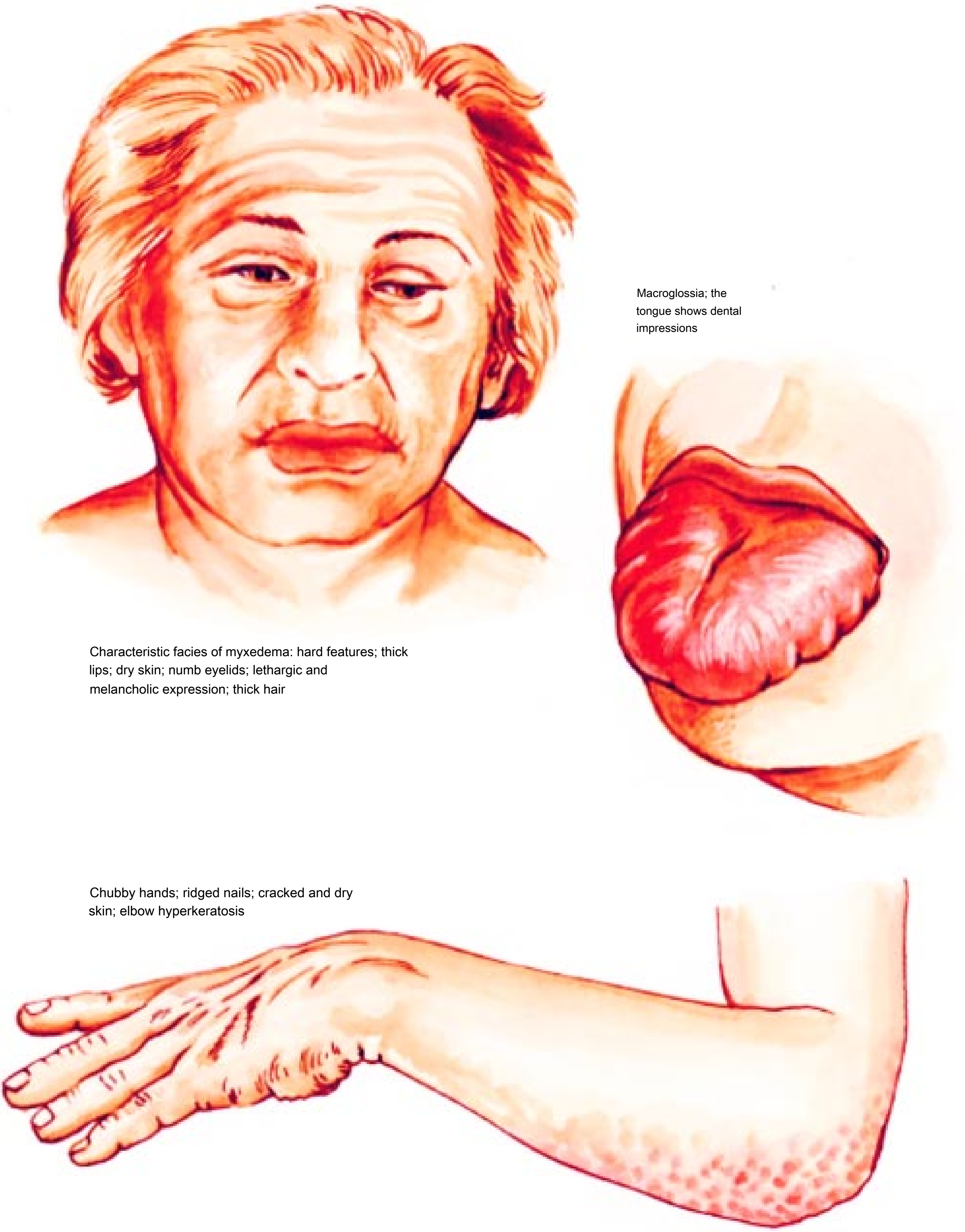
Nervous system

Headache is common, there may be dizziness or vertigo.


PRIMARY MYXEDEMA
PITUITARY MYXEDEMA
Differential features
Dry, brittle hair
Lethargy, lack of memory, slow cerebration (possible psychoses)
Edema of the face and eyelids
Thick tongue, slow speech
deep and harsh voice
Cold feeling
Reduced respiration
Enlarged heart, faint heart sounds, chest pain (occasional)
Hypertension (common)
Rough, dry, scaly, cold (follicular keratosis), yellowish skin (
slow pulse
Ascites
Menorrhagia (amenorrhea may be observed in the later stages of the disease)
Weakness
Reflexes, prolonged recovery
Finer and softer hair
Axillary weight loss
small heart
Hypotension
Less dry skin
not flaky
Pubic hair loss
Amenorrhea

PRIMARY MYXEDEMA	PITUITARY MYXEDEMA
T4 and T3: low: TSH: high	T4 and T3: low; TSH: decreased
PBI and BEI: low; do not increase after TSH	Low, but0 increase after TSH
^{131}: low 24-hour capture; does not increase after TSH	Low, but increases after TSH
Cholesterol: elevated (usually)	Normal (currently)

Fig.73.10 Hypothyroid syndrome.



Characteristic facies of myxedema: hard features; thick lips; dry skin; numb eyelids; lethargic and melancholic expression; thick hair

Macroglossia; the tongue shows dental impressions

Chubby hands; ridged nails; cracked and dry skin; elbow hyperkeratosis

Fig. 73.11 Myxedema facies. Alterations of the tongue and skin.

Patients are lazy, have bradypsychia (poor psychic and intellectual reaction), difficulty in associating ideas and appear clumsy in ordinary conversations.

They frequently present emotional changes, laughing and crying easily: affective clonus.

They have a tendency to drowsiness, they remain seated in any place for a long time and the ease with which they fall asleep is typical. Sometimes they present with nocturnal insomnia.

Although they are generally depressed, they may present symptoms of excitement, delirium and even olfactory, auditory and visual hallucinations; They can even reach a state of myxedema insanity.

In extreme cases, without treatment, these patients evolve towards a true coma, with hypoglycemia, bradycardia, hypothermia and sphincter incontinence.

With some frequency they present deafness that can be perceptual, conductive or mixed; In the study of the inner ear and the labyrinth, labyrinthine hyporeflexia is detected.

There is also vertigo, ringing in the ears, etc. The alteration of the visual organ is exceptional.

On physical examination they present decreased tendon, cutaneous and periosteal reflexes.

In many cases the diagnosis is confirmed by estimating the slow and prolonged relaxation of the Achilles tendon reflex: Achilles reflexogram.

digestive system

The symptoms originate from three pathophysiological mechanisms: infiltration of the wall of the digestive tract, difficulty in intestinal absorption and alterations of the neurovegetative system.

The following are common: anorexia, decreased salivary secretion, heavy digestion, persistent constipation, which are related to gastric hypochlorhydria and decreased intestinal motility.

circulatory system

The two most obvious findings are:

1. Vascular sclerosis, which occurs as a consequence of the alteration of lipid and cholesterol metabolism and is manifested clinically by cardiac anoxia secondary to coronary sclerosis and, also, by phenomena of peripheral deficit, including cerebral deficit.

2. Myxedema heart, which is extremely characteristic and originates from the degeneration of the myocardium with its infiltration.

Occasionally, the myxedema state leads to a true pericardial effusion. It is characterized by dyspnea on exertion and a sensation of cardiac oppression; The examination detects an enlarged heart that can affect all chambers. Bradycardia is constant. Blood pressure is normal, sometimes there is a slight decrease and only in cases with kidney involvement can it be high.

Heart tones are usually muffled.

Myxedema heart can produce true heart failure with its characteristic symptoms, which respond admirably to medication.

thyroid tion.

The ECG shows, as more typical, low voltage, bradycardia and a flattening of the T wave in all leads; QRS lengthening, notching, and decreased QT interval are less typical.

Respiratory system

Apart from the aphonia already described, catarrhal processes of the respiratory tract and bradypnea are common. It is not uncommon for them to have 8 or 10 breaths per minute.

The dyspnea that occurs is generally cardiac, although it may be due to secondary infections or pleural effusions.

locomotor system

Muscle or joint pain and cramps are common (in patients considered rheumatic). There is an evident decrease in segmental muscle strength. Myxedema infiltration gives the impression that the muscles are hypertrophic.

Alterations of the muscular system can present three modalities: simple muscle atrophy, a generalized or localized hypertrophic form or a third modality, which is the association of any of these forms with manifestations of myotonia. However, the most common is the presentation of pseudohypertrophy or true hypertrophy.

Bone alterations are characteristic of congenital and juvenile hypothyroidism.

Genitourinary system

There is decreased libido in both sexes with a tendency to impotence or frigidity and hypogonadism.

Generally there is a decrease in diuresis. Albuminuria is found in the urine, which sometimes acquires the character of a true lipoid nephrosis, which has given rise to the criterion of thyroid intervention in the pathogenesis of this disease.

In chronic cases there may be myxedema renal infiltration so intense that it causes true renal failure.

Neuroendocrine disorders

Together with myxedema and metabolic alterations, they constitute the pathophysiological tripod that explains all the symptoms.

The hypothalamus is especially affected, which explains some symptoms, such as feeling cold, drowsiness, apathy, affective clonus, psychosis, etc.

The pituitary gland may be inhibited in its gonadotropic, corticotropic and somatotropic hormones, which gives rise to developmental arrest, infantilism and decreased organic vital tone.

Thyrotropic hormone (TSH) may be decreased, when it comes to secondary (pituitary) hypothyroidism, or it may be increased, when the thyroid is primarily affected.

Due to a decrease in corticotropic hormone, a condition of hypocorticalism occurs with a decrease in urinary 17-ketosteroids.

Gonadotropin deficiency causes delay in gonadal development with a tendency to impotence, both psychological and

Complementary exams

1. Serum T4 and T3 are decreased.
2. TSH is elevated in the majority of cases that constitute thyroid-deprived or primary hypothyroidism; In the pituitary and hypothalamic, this hormone is decreased.
3. When the Achilles tendon reflex is examined, the reflexoachillogram is elongated.
4. The ECG shows microvoltage with lengthening of the PR and QT intervals, and inverted T waves.

In lipid metabolism we find an increase in lipemia, especially blood cholesterol.

In iodine metabolism there is, in addition to the decrease in T4 and T3 already mentioned, an intense decrease in the uptake of I'31 by the thyroid when this element is administered. In cases of primary hypothyroidism, there is hardly any uptake and it is almost completely eliminated in the urine. In secondary (pituitary) hypothyroidism, less is uptake than normal, but the normal figure is almost reached, which is 40% uptake. Protein iodine in the blood (PBI) is found to be decreased (normal figure: 3-7 gammas %) and has diagnostic value, although this investigation is no longer used if dosage of T4 and T3 is available.

In the hematological examination, anemia is present, almost always hypochromic; more rarely, hyperchromic type. The decrease in platelets is a fact on which much emphasis is placed, due to the tendency to metrorrhagia or other hemorrhagic expressions in hypothyroid patients. There is a tendency towards leukopenia with neutropenia and slight eosinophilia.

There is, finally, a decrease in the synthesis of vitamin A; In these cases, the conversion of carotenes to vitamin A does not occur in the liver.

Etiology (fig. 73.12)

Hypothyroidism in adults can occur due to the following causes:

1. Primary myxedema (see Fig. 73.10). It is the most frequent and interesting way; It is spontaneous, idiopathic, with no previous history. It is already considered a consequence of autoimmunization against thyroglobulin, which would chronically lead to the destruction of thyroid tissue. It is also known as Gull-Ord disease. Gull discovered the cretinoid state that usually occurs in adulthood in women, where it is more frequent 4:1 in relation to men. Ord called him myxedema, seeing the mucin infiltration of the skin.
2. Congenital enzyme defects. In the synthesis of thyroid hormone.
3. Serious voice deficiencies. In areas of endemic goiter.
4. Thyroid disease. There may be inflammation, thyroiditis (subacute and chronic), tumors, goiter, aplasia or atrophy.
5. Thyroidectomy. It rarely leads to myxedema, but if the excision is almost total, it appears as a late complication.
6. Radiotherapy and radioactive iodine. The application of these resources in hyperthyroidism can cause a myxedema state, if the dose is excessive.
7. Antithyroid drugs. Prolonged administration of these drugs can cause it.
8. Pituitary myxedema (see Fig. 73.10). Described in 1935 by Zondeck in a pituitary tumor. It is characterized by the fact that thyroid symptoms seem less and, on the other hand, there is a greater wealth of endocrine and hypothalamic symptoms, such as dysthermia, sleep disorders, etc.

   The functional tests of hypothyroidism that are altered return to normal when the patient is administered thyrotropic hormone, which does not occur if the hypothyroidism is primary.

   The hypothyroidism of Sheehan syndrome is also pituitary.
9. Hypothalamic myxedema. We mentioned it last; Complete cases have been published in which the lesion was in the hypothalamus.

Juvenile hypothyroidism

In reality it is nothing more than the hypothyroidism itself that we have described in adults, but with some differences. One of them is, that as presented in a

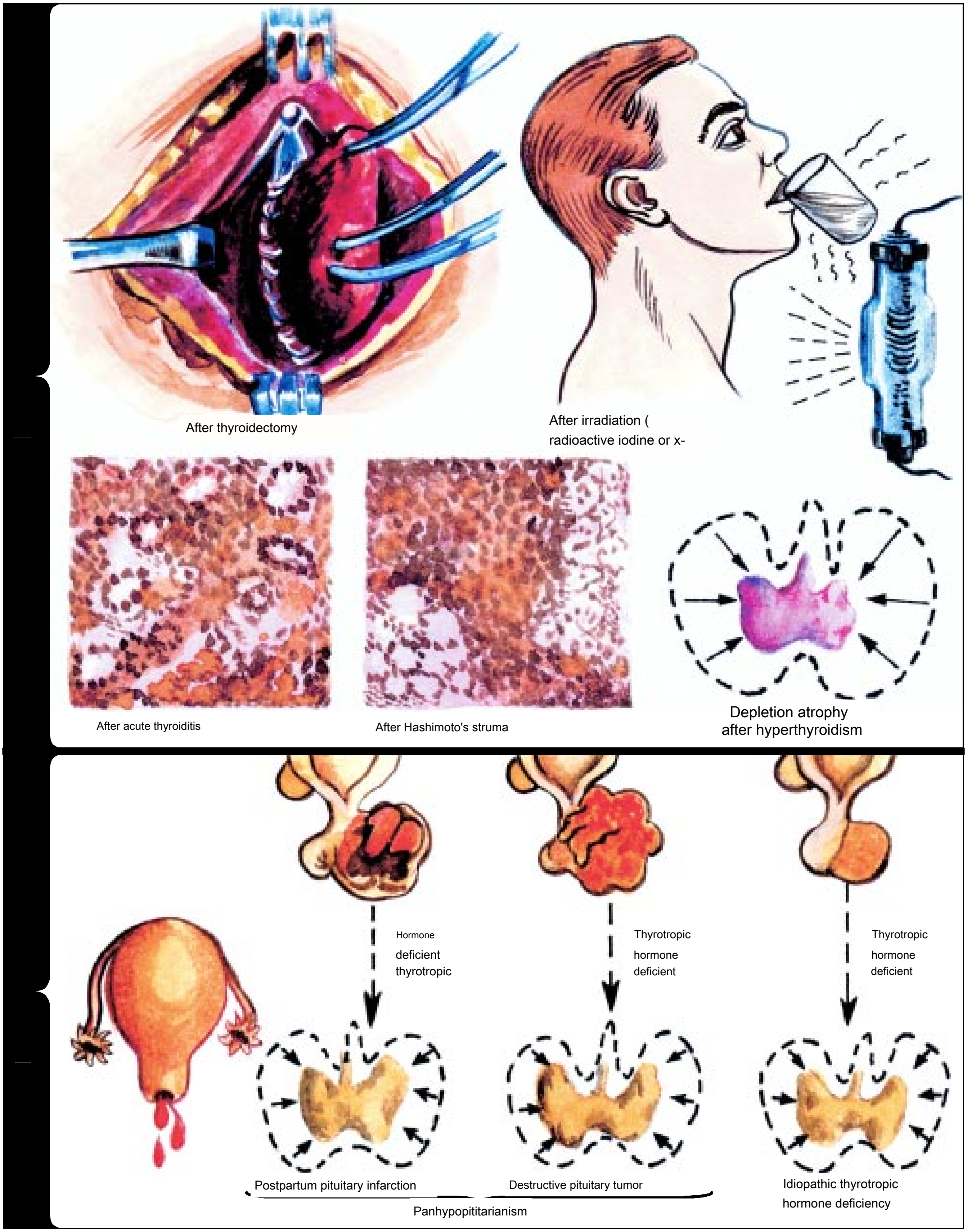

After thyroidectomy
After irradiation (
radioactive iodine or x-
After acute thyroiditis
After Hashimoto's struma
Depletion atrophy
after hyperthyroidism
Hormone
deficient
thyrotropic
Thyrotropic
hormone
deficient
Thyrotropic
hormone
deficient
Postpartum pituitary infarction
Destructive pituitary tumor
Panhypopititarianism
Idiopathic thyrotropic
hormone deficiency

Fig. 73.12 Etiology of adult myxedema.

age in which complete development has not occurred, it is affected and causes a delay in growth.

Another difference is that the nervous and psychointellectual alterations are very marked and psychic development and learning are manifestly diminished.

Associated with these disorders described, there is a delay in sexual development and puberty.

Myxedema is exceptional in juvenile hypothyroidism; it presents intermediate symptoms between adult hypothyroidism without myxedema and congenital hypothyroidism.

Congenital hypothyroidism (thyreosis) (see Fig. 66.15)

It is a thyroid insufficiency that generally occurs during fetal development and in some cases, early, in the first months of birth. In congenital cases there is no development of the thyroid body at all, there is thyroid aplasia, that is, true thyroidism or athyreosis. In early childhood there is a history of infections and poisoning.

Isolated sporadic or congenital cretinism occurs in healthy parents, who generally have had other children and only one is affected by the disease.

Endemic cretinism occurs in families or in so-called goiter endemic areas, almost always in mountainous areas with entire families of cretins, who coincide with other goiters.

Whatever the mechanism that generated it, when the thyroid hormone fails, the following appears:

1. The classic symptoms of hypothyroidism, although myxedema is generally absent.
2. Growth and development disorders that cause a marked height deficit. They are the so-called hypothyroid dwarfs, who give the impression of being children younger than their chronological age: they are rough, with very dry and rough skin, a very large tongue, limbs with short and wide fingers, with marked nervous disorders:

   psychic and intellectual poverty (true oligophrenics), with delay in speaking and walking. This marked state of mental and psychological deficit causes many authors to speak of hypothyroid idiocy or imbecility due to congenital thyroidism.

ENDOCRINOMETABOLIC SYSTEM. PARATHYROID AND PARATHYROID SYNDROMES

4

PARATHYROID ANATOMY (FIG. 74.1)

The parathyroid glands, located behind the thyroid, are normally four, one upper and one lower on each side of the neck; the upper two come from the right and left fourth gill slits, respectively; the lower pair, from the third cleft. In humans, accessory glands can form, from the cricoid cartilage to the mediastinum. There may be more than four; sometimes less.

Sometimes, the parathyroids are included in the thyroid parenchyma, which explains the danger of removing them together with the thyroid.

In infancy and early childhood, the parathyroids are made up of solid layers of principal cells. Each appears in the adult as a yellow-brown, oval, encapsulated nodule, weighing about 30 mg and measuring 4-6 mm in length, 2-4 mm in width and 0.5-2 mm in thickness.

These glands receive blood from the inferior thyroid artery.

PARATHYROID HISTOLOGY

They are basically made up of the so-called principal cells (small, dark). In the adult, two additional cell shapes are identified: the clear cell and the oxyphilic cell (granular and pink). The principal cell is the progenitor of these cells.

Despite biochemical studies and electron microscopy, it has been difficult to determine which type of cell produces parathyroid hormone (PTH). It is accepted that it can be produced by the main (active) cell, which has the ability to transform directly into the other two cell forms and that all cell types are related to hyperparathyroidism.

PHYSIOLOGY OF THE PARATHYROID (FIG.74.2)

The parathyroid glands regulate the metabolism of calcium (Ca) and phosphorus (P), therefore we must remember, even briefly, the concepts of the metabolism of these important minerals.

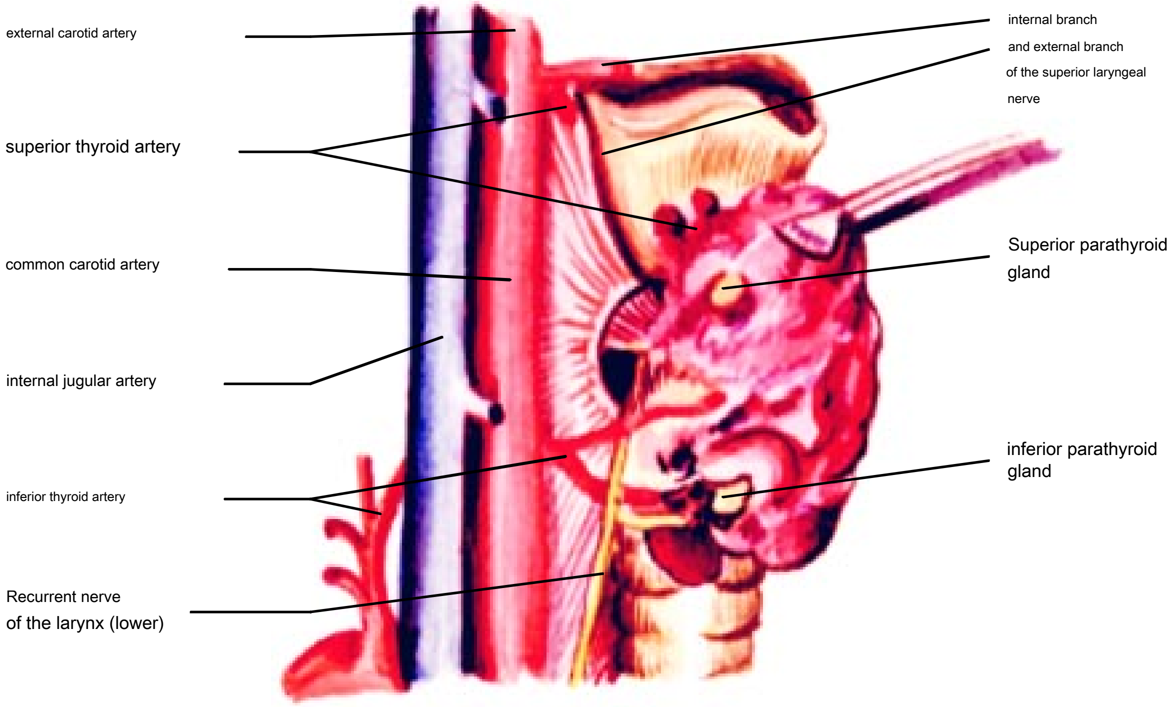


Fig. 74.1 Anatomy of the parathyroids.

The skeleton contains 99% of the total body Ca and 90% of the P. These minerals constantly circulate between the bone structure and the fluids and tissues, where they perform important vital functions.

CALCIUM

El Ca is essential for the formation of bones and teeth, as well as for blood coagulation. In addition, it regulates cell membrane permeability and neuromuscular excitability and maintains heart rate.

While insignificant changes in the concentration of Ca in the bones, and especially in the blood, produce serious organic disturbances, intense variations in the content of Ca in the blood and in the tissues determine only small disturbances.

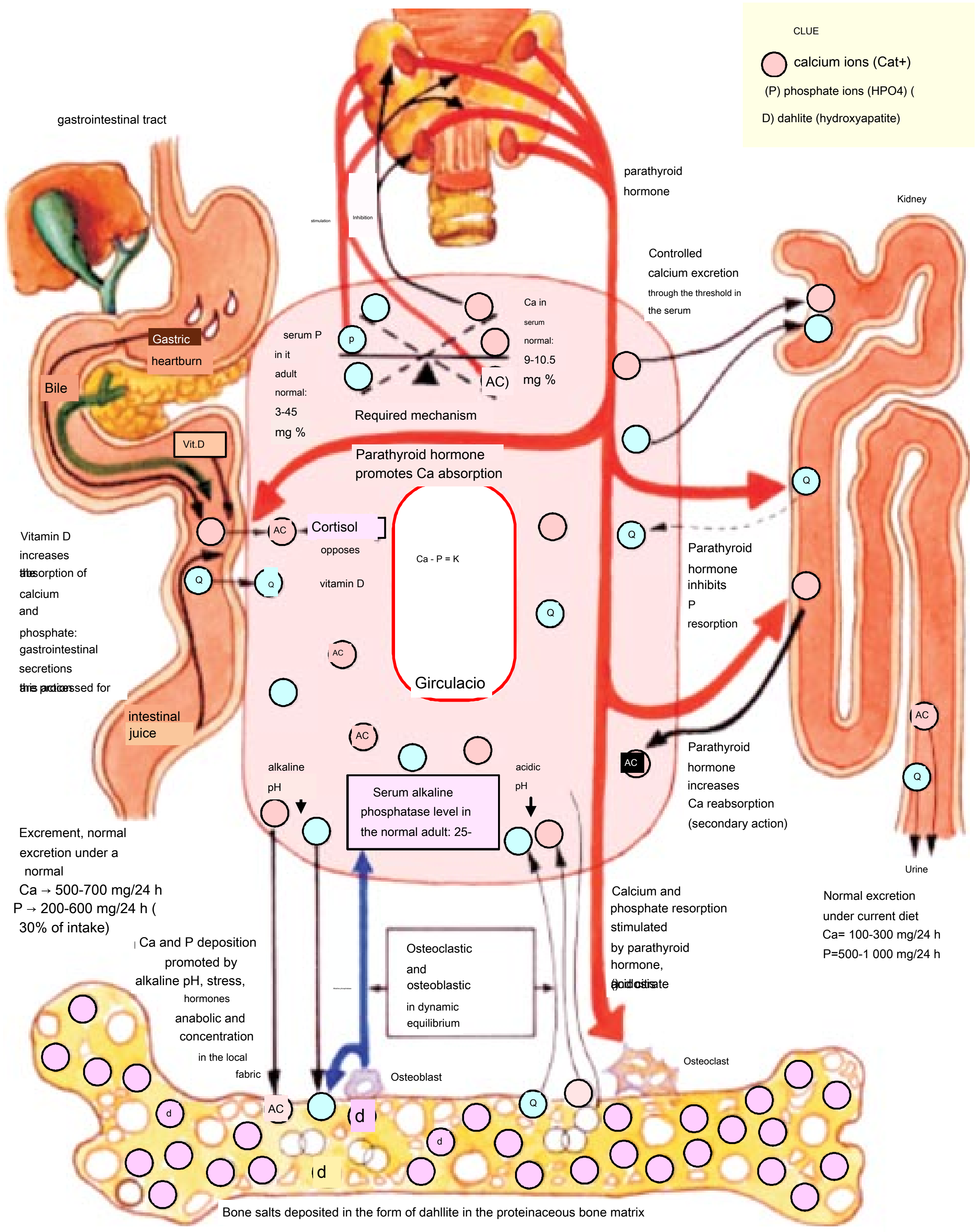
The daily demand for Ca (supplied especially by milk) varies from 0.9 g for adults to 2.5 g for children and lactating women. Ca is rapidly absorbed in the small intestine as long as there is an adequate amount of calciferol (vitamin D) or dihydrotachysterol (AT10), which is a derivative of irradiated ergosterol. The acidity of the gastrointestinal content facilitates the absorption of Ca; Fats and fatty acids that form insoluble soaps with Ca decrease it.

Absorbed Ca (about 100 mg per day) is deposited in the skeleton (osteoblastic activity) in increasing amounts throughout bone development, which begins at the end of fetal life. Osteoclasts (giant cells) that are at the sites of bone destruction remove Ca salts from the bone and release them into the interstitial fluid and plasma, where the concentration of Ca is rigidly maintained in proportions of 9-11.5 mg/dL (4.5-5.8 mEq/L). Part of this plasma or serum Ca is diffusible (4.5-6 mg/dL) in the form of ionized Ca; the rest, non-diffusible, is bound to proteins. Both fractions (under precise conditions of homeostatic regulation) are maintained in a rather unstable equilibrium, perhaps under the control of parathyroid hormone.

Insoluble Ca salts are excreted in feces (80%) and urine (20%). Excretion is regulated by factors such as the concentration of hydrogen ions, vitamins and hormones.

PHOSPHORUS

The daily demand for P is 1-1.25 g in adults; Children and pregnant and lactating women require higher amounts (2.5-3.9 g/day). The intestinal absorption of P from the diet (from milk, eggs, meat, cereals and others) is favored in the presence of dietary fats. Vitamin D slightly favors the absorption of P. This absorption is reduced by alkaline salts of metal cations (Ca, Fe or Al) that form insoluble phosphates with P. With the help of alkaline phosphatase, produced mainly by osteoblasts, most of the absorbed P is deposited in the bones. This phosphatase, activated by ascorbic acid and metal ions, hydrolyzes phosphates


CLUE
calcium ions (Cat+)
(P) phosphate ions (HPO4) (
D) dahlite (hydroxyapatite)
gastrointestinal tract
parathyroid
hormone
Kidney
stimulation
Inhibition
Controlled
calcium excretion
through the threshold in
the serum
Ca in
serum
normal:
9-10.5
mg %
serum P
in it
adult
normal:
3-45
mg %
p
AC)
Required mechanism
Gastric
heartburn
Bile
Vit.D
Parathyroid hormone
promotes Ca absorption
AC
Cortisol
opposes
vitamin D
Q
Ca - P = K
Girculacio
Vitamin D
increases
the absorption of
calcium
and
phosphate:
gastrointestinal
secretions
this process for
intestinal
juice
Parathyroid
hormone
inhibits
P
resorption
Parathyroid
hormone
increases
Ca reabsorption
(secondary action)
alkaline
pH
acidic
pH
Serum alkaline
phosphatase level in
the normal adult: 25-
Excrement, normal
excretion under a
normal
Ca → 500-700 mg/24 h
P → 200-600 mg/24 h (
30% of intake)
Ca and P deposition
promoted by
alkaline pH, stress,
hormones
anabolic and
concentration
in the local
fabric
Osteoclastic
and
osteoblastic
in dynamic
equilibrium
Calcium and
phosphate resorption
stimulated
by parathyroid
hormone,
acidosis citrate
Urine
Normal excretion
under current diet
Ca= 100-300 mg/24 h
P=500-1 000 mg/24 h
Osteoblast
Osteoclast
d
Bone salts deposited in the form of dahllite in the proteinaceous bone matrix

Matrix proliferation requires protein, vitamin C, anabolic homones (androgens, estrogens, STH plus mobility stress.

Matrix resorption is favored by catabolic hormones (11-oxysteroids/cortisol/thyroid) parathyroid hormone plus immobilization

Fig. 74.2 Biological actions of parathyroid hormones.

organics releasing inorganic P, part of which will be incorporated directly into the bones, blood and tissues. The rest of the P remains in the blood plasma: inorganic P 3-4.6 mg/dL.

60% of P is excreted in urine and 40% in feces. The same factors that regulate Ca metabolism intervene in P excretion.

PARATHYROID HORMONE

It is the main regulator, but not the only one, of serum calcium concentration, since calcitonin and vitamin D also participate.

Parathyroid hormone is a polypeptide of 84 amino acids. The complete structure of bovine and porcine hormones is known, and part of the sequence of human parathyroid hormone.

The biosynthesis of the hormone is carried out from a prohormone molecule of 90 amino acids.

Inside the gland it is transformed into a polypeptide of 84 amino acids that, for some, is first stored and secreted later, according to physiological stimuli.

Others consider that it is synthesized and secreted continuously.

Radioimmunological studies demonstrate that calcium is the main factor in the control of hormone secretion. If the concentration of blood calcium decreases, hormonal secretion occurs that accelerates the transfer of calcium from the bones and the glomerular filtrate to the extracellular fluids, also increasing the urinary elimination of phosphate by direct effect on the kidney.

The action of paratormone is carried out on 3-5'-cyclic andenosine monophosphate (3'5-cAMP), which is produced by direct specific hormonal activation of adenyl cyclase, in the kidney and bone. The resulting increase in the concentration of 3'5-cAMP in the renal cortex and bone activates other intracellular processes that explain the physiological effects of the hormone. This is also verified, since there is an increased urinary excretion of 3'5-cAMP.

CALCITONIN

It is the other polypeptide hormone that participates in the regulation of calcium and bone metabolism.

Calcitonin is secreted by the parafollicular cells of the thyroid and is therefore also called thyrocalcitonin. Its secretion rate is related to the concentration of calcium in the plasma. The hypocalcemic effect of calcitonin is carried out, probably due to inhibition of the action of parathyroid hormone on bone resorption and its physiological action is apparently due to the adenylate and cAMP systems.

The structure of calcitonin is known, which has allowed the synthesis of this hormone. Its evaluation is done by radioimmunological methods.

VITAMINAD, 0 CHOLECALCIFEROL

Observations made raise whether the role of parathyroid hormone is measured by the metabolites of vitamin Dosi acts, directly, in harmony with these metabolites.

Cholecalciferol is synthesized in the skin under the action of photons of ultraviolet light, it is activated in the liver and subsequently in the kidney, forming 1,25-dihydroxycholecalciferol, a steroid hormone that acts powerfully on two target organs: the mucosa of the small intestine, where it induces the synthesis of proteins that ensure the transport of calcium from the intestinal lumen to the body, and the osteocytes, where its action is reinforced. and joins that of the parathyroid hormone.

It is still difficult to separate the direct and indirect reactions of parathyroid hormone and vitamin D on the bones, kidneys or intestines; Future observations will give rise to new concepts in calcium regulation.

In summary, we can say that the effects of the increase in the concentration of parathyroid hormone

Are:

- Increase in serum calcium concentration.
- Increased absorption of dietary calcium by the intestine.
- Increased excretion of calcium in the urine with possible urolithiasis, nephrocalcinosis, as well as secondary changes.
- Release of calcium from its main deposit (skeleton), which causes bone resorption and bone disease.
- Conversely, the decrease in the concentration of parathyroid hormone produces hypocalcemia that can cause the clinical syndrome of tetany.

MAGNESI0

We will point out the role of magnesium in the action of parathyroid hormone. Hypomagnesemia also stimulates the secretion of parathyroid hormone.

PARATHYROID SYNDROMES

Preliminary considerations

To study parathyroid syndromes, it is advisable to consider the pathology in terms of function disorders, although there are non-functional lesions of the parathyroid glands (tumors and cysts), but of secondary importance and frequency.

Hyperparathyroidism may exist as a primary disease when increased amounts of parathyroid hormone are produced by hyperplastic or neoplastic glands (adenoma and carcinoma) or when non-endocrine tumors secrete parathyroid hormone or a biologically similar product.

Secondary hyperparathyroidism results whenever there is some cause of chronic hypocalcemia, as occurs in chronic renal failure, alimentary rickets, and intestinal malabsorption syndromes.

Parathyroid hyperfunction is a compensatory reaction in these cases. It is sometimes suggested that some primary hyperplasias or functioning adenomas are actually responses to ill-defined stimuli and, consequently, forms of secondary hyperparathyroidism.

To produce hyperfunction, an unknown or undiscovered stimulus causes a proliferative response in the form of hyperplasia. Since the stimulus is unknown, we call it primary hyperplasia.

Over time, continued proliferation within the glands could cause adenoma and even carcinoma. This would explain the cases of glandular hyperplasia with one or more adenomas found in patients with apparent secondary hyperparathyroidism, who in the course of the chronic condition have presented true functional adenomas (fig. 74.3).

The schematic separation between secondary and primary hyperparathyroidism and the separation of the morphological reactions in the latter (hyperplasia, adenoma and carcinoma) are somewhat arbitrary divisions.

The transformation of secondary hyperparathyroidism into autonomic hyperfunction is currently called tertiary hyperparathyroidism.

Hypoparathyroidism is largely a functional metabolic disorder that is accompanied by nonspecific anatomical changes, except those related to the parathyroid glands.

We mention the pseudohypoparathyroidism syndrome described by Albright and collaborators in 1942 here to point out that, although there are biochemical alterations of hypocalcemia and hyperphosphatemia with low calcium elimination, the effector tissues do not respond to the hormone. It is currently considered a familial disorder, due to the inability of the end organ to respond to the hormone, which is actually secreted in excessive quantities.

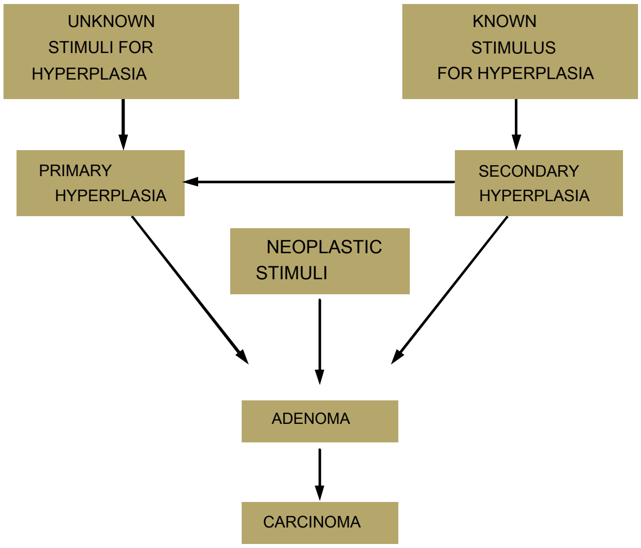


Fig. 74.3 Scheme of the relationships between hyperplastic and neoplastic parathyroid states.

Medullary thyroid carcinoma is a complex pathophysiological disorder in which the tumor secretes excessive amounts of calcitonin with elevated blood levels corresponding to it.

In some cases of this syndrome, primary hyperparathyroidism, pheochromocytoma, Cushing's syndrome, or multiple associated neuromas are

PRIMARY HYPERPARATHYROID SYNDROME

Concept

It is the clinical syndrome produced by excessive and poorly regulated secretion of parathyroid hormone. Hypercalcemia, osteitis fibrosa cystica and tissue calcification are the consequences of the excessive effect of the hormone on the bones and kidney.

Syndromogenesis or pathophysiology (ig. 74.4)

Excessive production of parathyroid hormone leads to hypercalcemia due to increased stimulation of osteoclastic activity of the bone, which causes the release of calcium and phosphate. Calcium absorption from the intestine and calcium reabsorption by the renal tubule are also increased. Similarly, parathyroid hormone inhibits tubular phosphate reabsorption (or promotes tubular secretion of phosphate) causing excessive loss of phosphate in the urine. Through their reciprocal interaction, decreasing serum phosphorus level raises serum calcium level. The final effect of these chemical changes is an increase in serum calcium and a decrease in serum phosphate, with an increase in the amounts of calcium and phosphate eliminated in the urine.

This predisposes to the formation of calcium phosphate and calcium oxalate kidney stones.

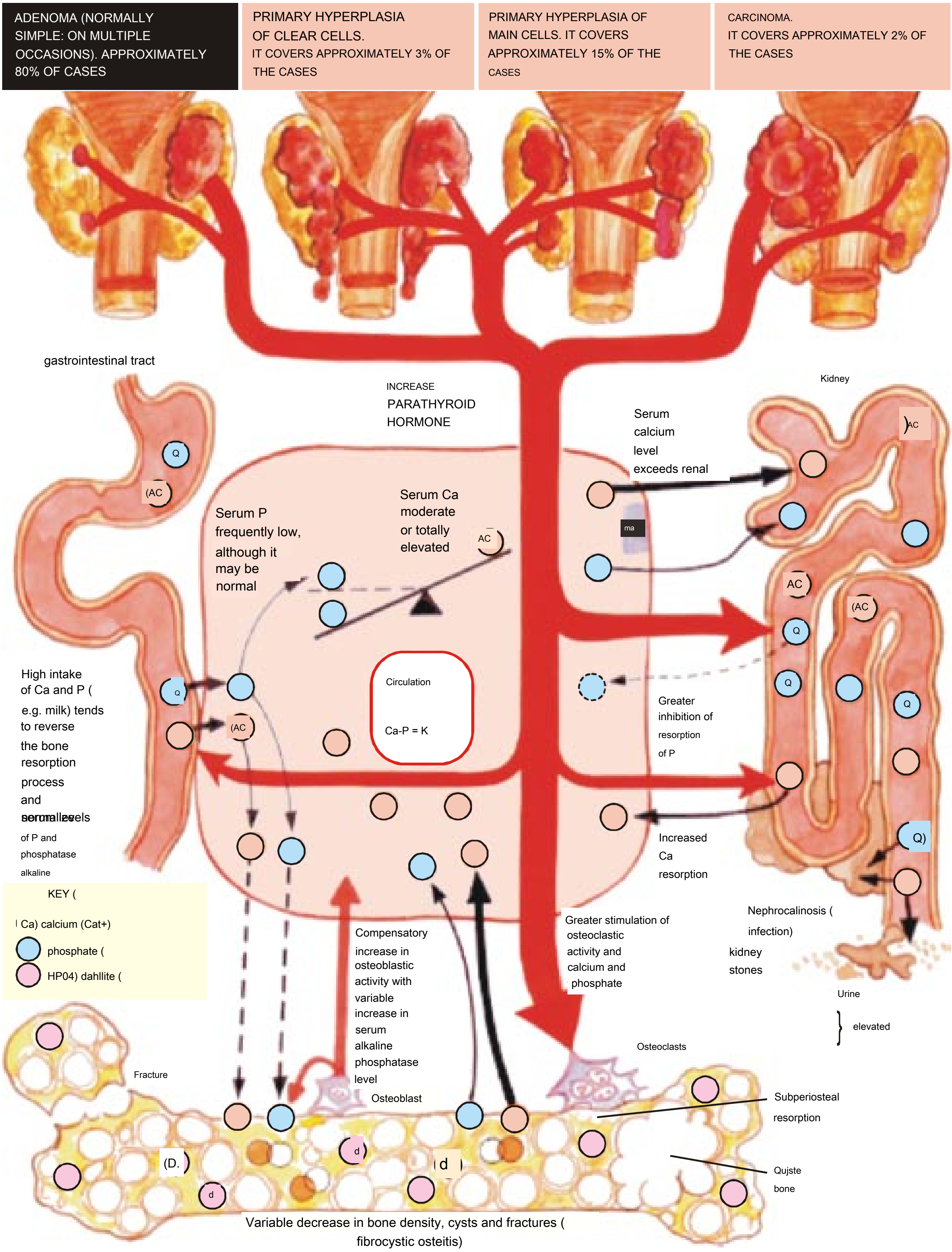

ADENOMA (NORMALLY SIMPLE: ON MULTIPLE OCCASIONS). APPROXIMATELY 80% OF CASES
PRIMARY HYPERPLASIA OF CLEAR CELLS. IT COVERS APPROXIMATELY 3% OF THE CASES
PRIMARY HYPERPLASIA OF MAIN CELLS. IT COVERS APPROXIMATELY 15% OF THE CASES
CARCINOMA. IT COVERS APPROXIMATELY 2% OF THE CASES
gastrointestinal tract
INCREASE PARATHYROID HORMONE
Kidney
Serum calcium level exceeds renal
ma
Q
(AC
Serum P frequently low, although it may be normal
Serum Ca moderate or totally elevated
AC
High intake of Ca and P (e.g. milk) tends to reverse the bone resorption process and normalizes serum levels of P and phosphatase alkaline
Circulation
Ca-P = K
Greater inhibition of resorption of P
Increased Ca resorption
KEY (
Ca) calcium (Cat+)
phosphate (
HP04) dahllite (
Compensatory increase in osteoblastic activity with variable increase in serum alkaline phosphatase level
Greater stimulation of osteoclastic activity and calcium and phosphate
Nephrocalinosis (infection) kidney stones
Urine
elevated
Osteoclasts
Fracture
Osteoblast
Subperiosteal resorption
Qujste bone
(D.
d
Q)
Variable decrease in bone density, cysts and fractures (fibrocystic osteitis)

Fig. 74.4 Pathophysiology and etiology of primary hyperparathyroidism.

Calcium precipitation may sometimes occur in the kidney tissue, causing nephrocalcinosis and often associated with infection. In 25% of cases of primary hyperparathyroidism, clinical evidence of bone disease, marked bone resorption, compensatory increase in osteoblastic activity and increase in the serum level of alkaline phosphatase are observed. This can be evidenced radiologically by subperiosteal resorption changes, decreased bone density, cyst formation, and sometimes even fractures (osteitis fibrosa cystica).

Pathophysiological alterations mainly lead to stone formation in 70% or more of cases and to bone disease in only approximately 25% of them.

Syndromography or positive diagnosis

Clinical picture (fig. 74.5)

Some cases of hyperparathyroidism are discovered in systematic clinical studies, when hypercalcaemia is found in patients undergoing laboratory studies or more precise investigations.

Sometimes it is a mild bone disease with hypercalcemia. In other cases there is polyuria and polydipsia, and when investigating in the clinic we find kidney stones, hypercalcemia and hypophosphatemia.

In other patients, the disease follows an insidious course, sometimes for years, and the symptoms may be urinary, especially in the form of renal colic.

In a third group we find the syndrome already established.

General symptoms

There is asthenia, easy fatigue and weight loss.

Digestive symptoms

Epigastric pain, anorexia, nausea, vomiting, and constipation are often present. There is a predisposition to gastroduodenal ulcer and sometimes pancreatic symptoms due to calcinosis.

Symptoms of bone tissue

Bone pain, existence of easy fractures, bone swellings or tumors, bone deformities and alterations such as diffuse and generalized bone hypertrophy or localized to certain bones that are accessible to palpation. Hippocratic or clubbing fingers may be found. Sometimes, a picture of gout or pseudogout.

Urinary symptoms

For some, they constitute the fundamental and often revealing symptomatology. It can take two types:

1. Calcium nephrolithiasis, due to the precipitation of excess calcium with repeated stone formation and its classic symptoms, including renal colic.
2. Nephrocalcinosis, with calcium deposits in the renal parenchyma.

Sometimes true calcium diabetes occurs with polyuria, polydipsia and increased calcium elimination.

Cardiovascular symptoms

The symptoms are due to arterial calcifications (especially in the tunica media) including coronary calcifications with their sequelae.

Calcifications can affect the myocardium, with alterations in cardiac conduction.

The ECG shows a shortening of the QT interval.

A hypertensive syndrome may be associated.

Respiratory symptoms

They occur as an expression of calcification in the bronchi and lungs.

Neuromuscular symptoms

There is muscle and igament hypotonia, with hyporeflexia, asthenia and muscle atrophy, and muscle calcifications. Muscular asthenia is sometimes so evident that it raises the suspicion of Addison's disease or severe hypokalemia.

Electrical examination indicates hypochronaxia and hypoexcitability to direct galvanic current.

Psychic alterations

There is confusion, apathy and lethargy.

Genital symptoms

Gonadal insufficiency and early menopause may coexist.

Eye symptoms

Long-lasting hypercalcemia can produce ocular calcifications that manifest as band keratopathy, the appearance of opaque substance in vertical lines, parallel to and within the sclerocorneal limbus; or in the form of conjunctival crystals outside the limbus and under the eyelids. The examination should be done with a slit lamp.

Primary hyperparathyroidism syndrome may be part of a multiple endocrine adenomatosis syndrome.

In this case, the concomitant pituitary and pancreatic tumors produce associated endocrinopathies: hypopituitarism, peptic ulcer due to excessive gastrin secretion (Zollinger-Ellison syndrome) or hypoglycemia due to insulin secretion.

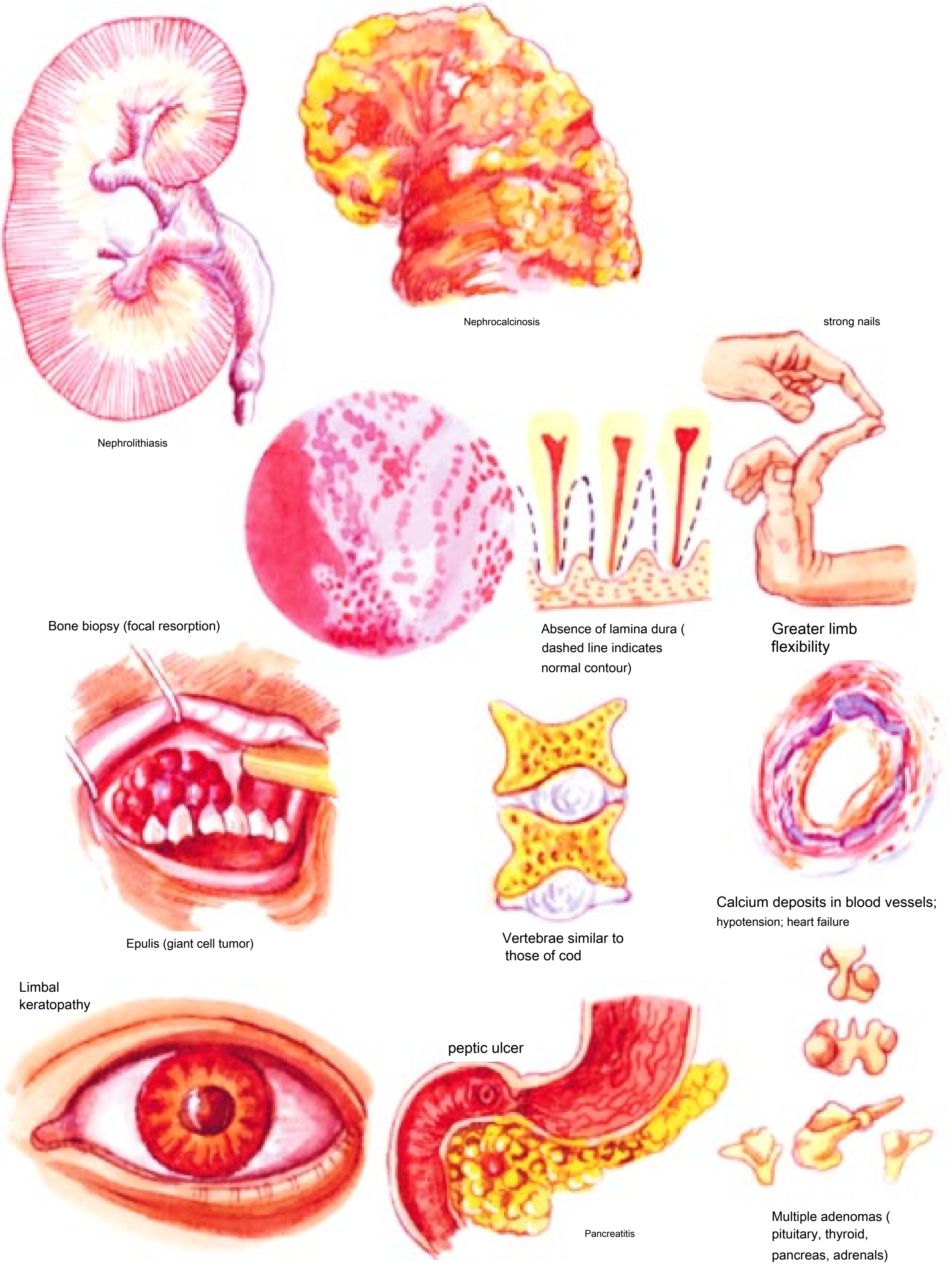

Nephrocalcinosis
strong nails
Nephrolithiasis
Bone biopsy (focal resorption)
Absence of lamina dura (dashed line indicates normal contour)
Greater limb flexibility
Calcium deposits in blood vessels;
hypotension; heart failure
Epulis (giant cell tumor)
Vertebrae similar to those of cod
Limbal keratopathy
peptic ulcer
Pancreatitis
Multiple adenomas (pituitary, thyroid, pancreas, adrenals)

Fig. 74.5 Clinical picture of hyperparathyroidism.

There may be another type associated with endocrinopathies, which includes pheochromocytoma, parathyroid adenoma, and medullary thyroid carcinoma.

These two types can be hereditary.

Complementary exams

1. Hypercalcemia is almost always detected in primary hyperparathyroidism (in 50%). The elimination of calcium within 24 hours (calciuria) is high.
2. Plasma alkaline phosphatase is obviously elevated when there is bone disease.
3. There is an increase in the urinary elimination of hydroxyproline and
4. Hypophosphoremia below 2.5 mg/dL is a major sign when it exists, but unfortunately it is inconsistent, as it is sometimes masked by renal failure. Tubular phosphate reabsorption is decreased.
5. The ionogram demonstrates metabolic acidosis with hyperchloremia and low serum bicarbonate concentration in 25% of patients.
6. Sometimes we find hypokalemia and hypomagnesemia.
7. Anemia with accelerated erythrocyte sedimentation rate is frequently present.
8. In special laboratories, the urinary excretion of 3'5'-cAMP can be determined. In primary hyperparathyroidism, the mean urinary excretion of 3'5-cAMP increases and, conversely, in hypoparathyroidism, excretion is low. The same occurs with certain non-parathyroid cancers that are accompanied by
9. Assessment of parathyroid hormone by radioimmunological methods in special laboratories. Hypercalcemia unrelated to parathyroid hormone is required by this method, since in cases of hyperparathyroidism, high concentrations of the hormone are demonstrated, in relation to the calcium content in the plasma.
10. Skeletal alterations can be specified with the radiological study. In addition to the initial osteoporosis, a series of histological alterations are added, such as necrosis, fibrous invasion in the osteolytic areas, giant cells, lymphocytes, with the characteristic radiological alterations: sometimes the bone appears as flocculated or moth-eaten and in others, as with cysts like pseudotumors.

    Depending on whether osteoporosis or cysts predominate, the clinical forms of the syndrome will be defined.
11. Tubular calcium reabsorption is increased and renal phosphate clearance is increased.
12. The corticosteroid test rules out hyperpartthyroidism when hypercalcemia is inhibited.

Etiology (er fig. 74.4)

Primary hyperparathyroid syndrome can be due, as we have pointed out, to primary hyperplasia of the cells, single or multiple adenomas, including adenomas of other glands (multiple endocrine adenomatosis) or glandular carcinoma.

In an analysis of 343 cases, Cope (1966) reported the following injuries:

	Percentage
Single adenoma	76
Double adenoma	4
Carcinoma	
Clear cell hyperplasia Principal (dark) cell hyperplasia	4
	12

It does not mention cases of hyperplasia and mixed adenomas, or hyperparathyroidism caused by non-endocrine tumors, which are increasingly observed. In this group, undifferentiated lung carcinoma and renal cell carcinoma top the list. Retroperitoneal sarcoma and liver metastases from breast carcinoma have also been reported.

In these cases that are part of the so-called paraneoplastic syndromes, the secretion product of these tumors, although it has sensitizing activity, is not structurally identical to the normal parathyroid hormone.

evil.

Other authors point out a different figure for different etiologies.

Secondary hyperparathyroidism appears as a compensation mechanism in disorders that produce true hypocalcemia: hypovitaminosis D, rickets in relation to vitamin D, malabsorption syndromes with calcium loss and in chronic renal failure with hyperphosphatemia and renal tubular acidosis.

Families suffering from autosomal dominantly inherited hyperparathyroidism have been reported.

HYPOPARATHYROID SYNDROME

Concept

It is the syndrome characterized by deficient secretion of parathyroid hormone with consequent hypocalcemia.

Syndromogenesis or pathophysiology (fig. 74.6)

The physiological consequences of insufficient parathyroid hormone secretion are: low calcium levels,

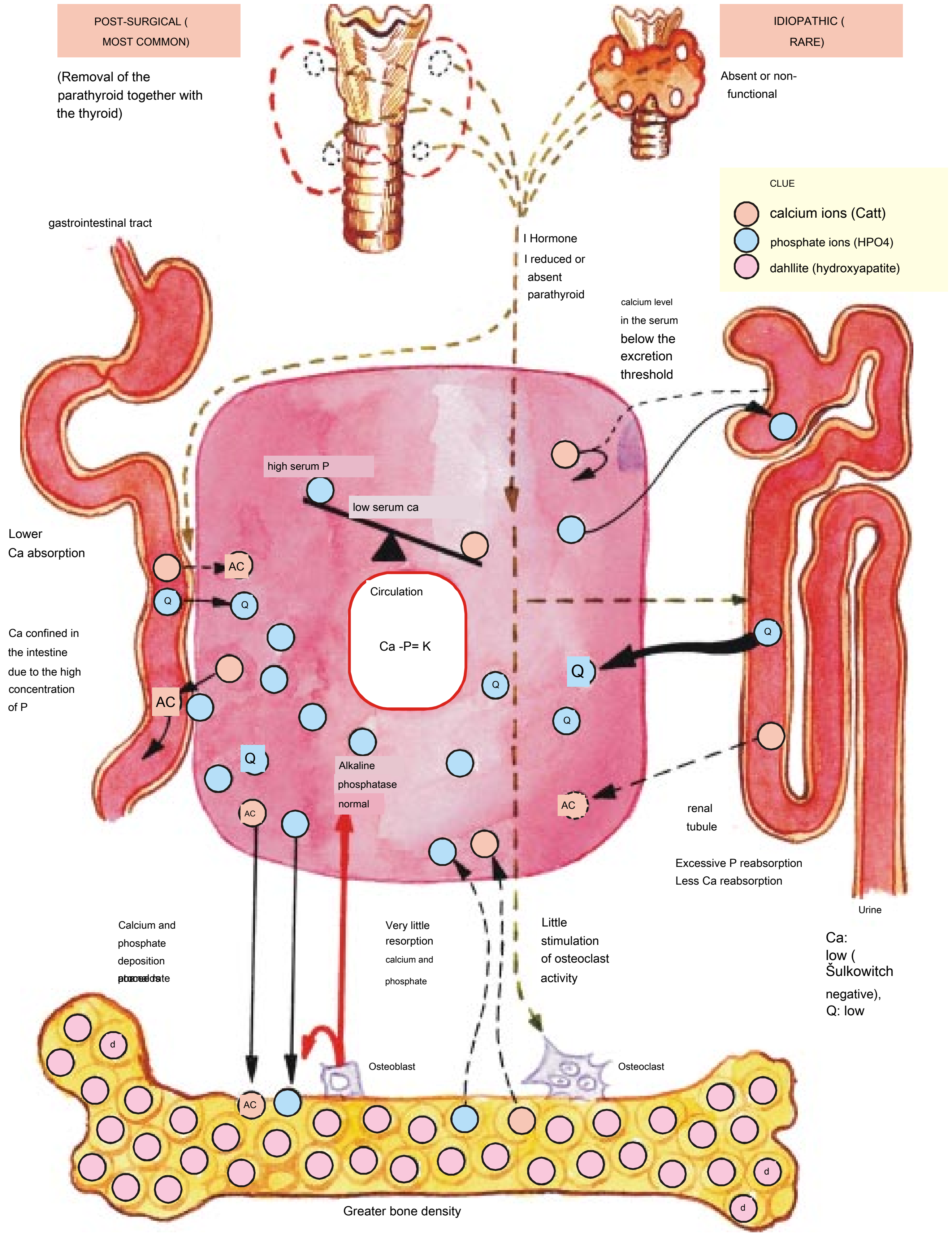

POST-SURGICAL (
MOST COMMON)
(Removal of the parathyroid together with the thyroid)
IDIOPATHIC (
RARE)
Absent or non-functional
CLUE
calcium ions (Catt)
phosphate ions (HPO4)
dahllite (hydroxyapatite)
gastrointestinal tract
I Hormone
I reduced or absent parathyroid
calcium level in the serum below the excretion threshold
high serum P
low serum ca
Lower Ca absorption
AC
Q
Circulation
Ca -P= K
Ca confined in the intestine due to the high concentration of P
Alkaline phosphatase normal
renal tubule
Excessive P reabsorption
Less Ca reabsorption
Urine
Ca: low (Šulkowitch negative), Q: low
Calcium and phosphate deposition
proceeds
phosphate
Very little resorption calcium and phosphate
Little stimulation of osteoclast activity
Osteoblast
Osteoclast
d
Greater bone density

Fig. 74.6 Pathophysiology of hypoparathyroidism.

high phosphatemia and decreased urinary phosphate clearance. Hypocalcemia is accompanied by low calcium concentration in the urine (hypocalciuria). Calcium clearance in the urine is almost zero when calcium levels decrease below 7 mg/dL.

Hypocalcemia is an expression of poor bone resorption due to parathyroid deficiency.

The decreased ionic calcium concentration in the extracellular fluid increases neuromuscular excitability and produces tetany, with its symptomatic process. Hyperphosphatemia is due to the absence of the hormonal effect on phosphate clearance by the kidney. The elimination rate of 3'5-cAMP is also decreased, but, unlike what occurs in pseudohypoparathyroidism, the response to exogenous parathyroid hormone is normal.

Syndromography or positive diagnosis

Clinical picture (fig. 74.7)

It depends fundamentally on the low concentration of calcium in the extracellular fluid.

General symptoms

Sometimes they suffer from a feeling of fatigue, asthenia, weight loss, anorexia; other times, erratic rheumatic-type pains.

Bone alterations

In children, disorders of bone and dental growth can be observed, with typical teeth showing alterations in the enamel, such as spots and grooves or horizontal bands and, in addition, serrated or serrated contours. In adults, because the dentition and skeleton are fully developed, these alterations do not occur.

Trophic alterations

The skin of these patients can become rough, dry, rough, reminiscent of the skin of patients with hypovitaminosis A or hypothyroid patients.

The hair is fragile, sparse and thin, it falls out easily; eyebrows, armpit and pubic hair are weak. The nails are fragile, brittle, with striations. Moniliasis is common in idiopathic hypoparathyroidism.

Very typical cataracts can occur in the ocular lens, due to a calcium deposit in its structure.

Alterations in the cardiovascular system

Symptoms of cardiovascular erethism appear: palpitations, sensation of oppression and sometimes, symptoms of coronary insufficiency.

The ECG shows a lengthening of the QT interval that may be associated with flattened T.

Vascular spasms with paleness, coldness and sometimes peripheral edema are common.

Alterations in the digestive system

There are dyspeptic manifestations, with flatulence and vomiting; Sometimes abdominal cramps occur associated with a diarrheal syndrome. Sometimes constipation predominates. Abdominal pain can be so intense that one thinks of an acute abdomen.

Alterations in the nervous system

The following are observed: headaches, vertigo and various pains, sometimes in certain muscular territories. There may be visual and auditory alterations, paresthesias, especially typical in the ulnar territory. Neurovegetative signs may occur with sweating, a feeling of coldness and hot flashes. On examination, osteotendinous hyperreflexia is common.

The psychic state shows great excitability that is not constant; Sometimes they are phases of depression, with a very volatile temperament or character and, sometimes, it reaches a true psychotic syndrome.

The EEG indicates slow waves, isolated or in series, with isolated needles, which are produced by hypocalcemia, since the intravenous injection of calcium normalizes them.

Papilla edema, which may be associated with increased intracranial pressure, accompanies hypocalcemia and is normalized by correction (Fig. 74.8).

Tetany syndrome

The most characteristic condition from a clinical point of view is tetany; however, it can take two forms: latent tetany and manifest tetany (see Fig. 74.8).

In latent tetany, the pathophysiological state is characterized because crises only occur in the face of circumstantial stimuli or those provoked by the doctor, who recognizes this state of neuromuscular excitability through examination and clinical maneuvers, as we will see later.

Overt tetany is that clinical form of hypoparathyroidism that eventually produces crises or attacks of spontaneous tetany or spasmophilia.

Within the clinical picture of the syndrome, an acute and a chronic form are described, according to the intensity and acuity of the presentation of the picture.

latent tetany

Latent tetany can be demonstrated by compressing the middle part of the arm with a rubber band or blood pressure cuff; then it is observed that the fingers of the hand are placed in hyperextension and the finger

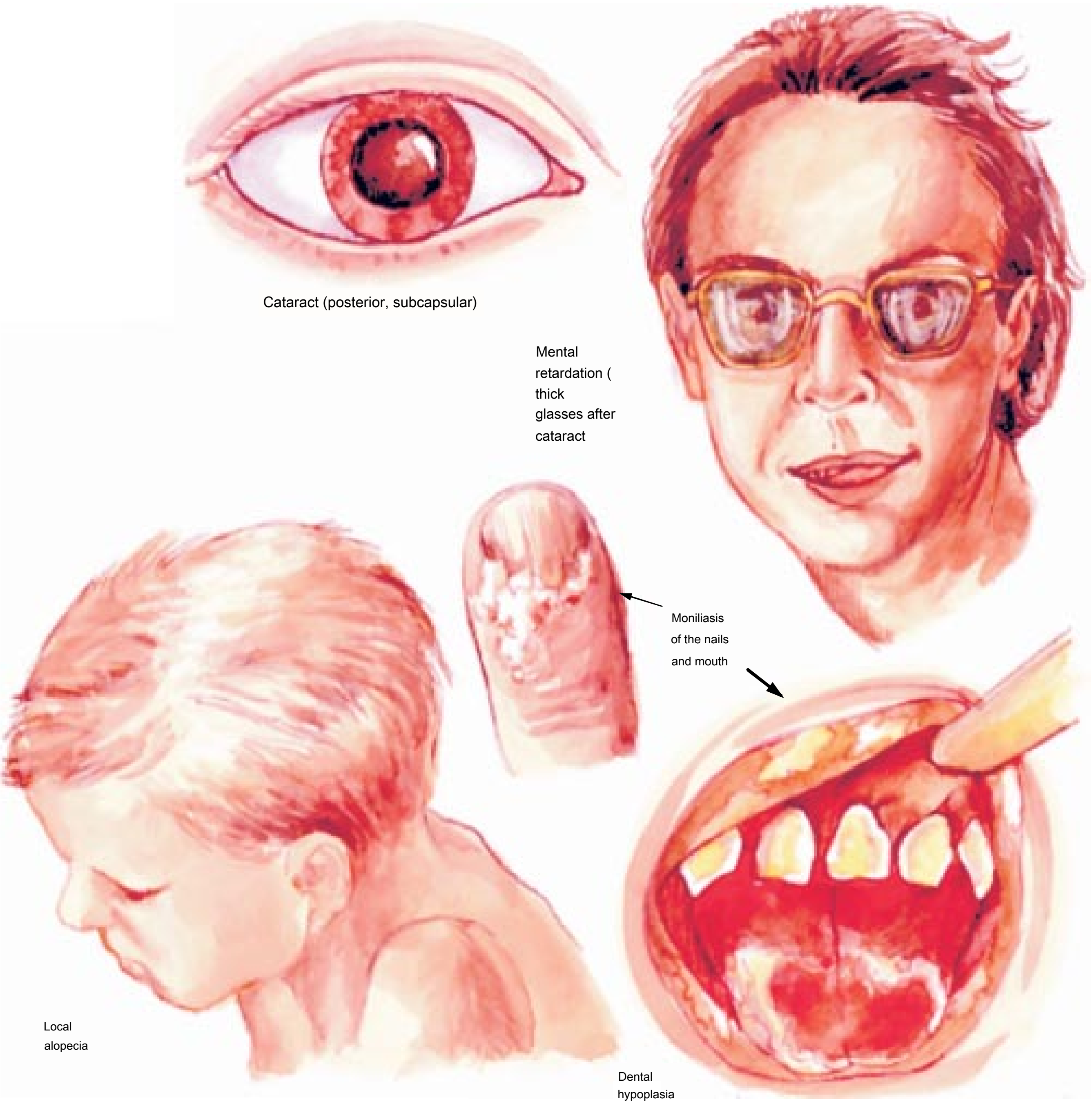

Cataract (posterior, subcapsular)
Mental retardation (thick glasses after cataract
Moniliasis of the nails and mouth
Local alopecia
Dental hypoplasia

Fig. 74.7 Clinical picture of chronic hypoparathyroidism.

thumb in forced adduction; at the same time the palm of the hand cups, this has been called the midwife's hand (Trousseau's sign) (fig. 74.9). This sign is spontaneously observed in attacks of acute tetany.

Sometimes when this sign is investigated on one side, it is observed on the other hand (Frankl Hochwart sign) or vascular spasm and anemia of the fingers may be added (Khan and Falta sign).

Percussing the temporal branch of the facial nerve causes contraction of the orbicularis, superciliary and frontal muscles (Weiss sign) (Fig. 74.10).

By striking the middle of a line that extends from the external auditory canal to the oral commissure, contraction of the facial muscles is obtained (Chvostek's sign) (fig. 74.11).

Percussion of the labial commissure causes propulsion of the lips forward (Escherich's sign) (Fig. 74.12) and sometimes there are seizures of the entire face (Uffenheimer's facies). This mechanical hyperexcitability can also be caused in other nerve trunks: in the peroneus (Lust's sign) and in the tibia (Schlesinger's sign).

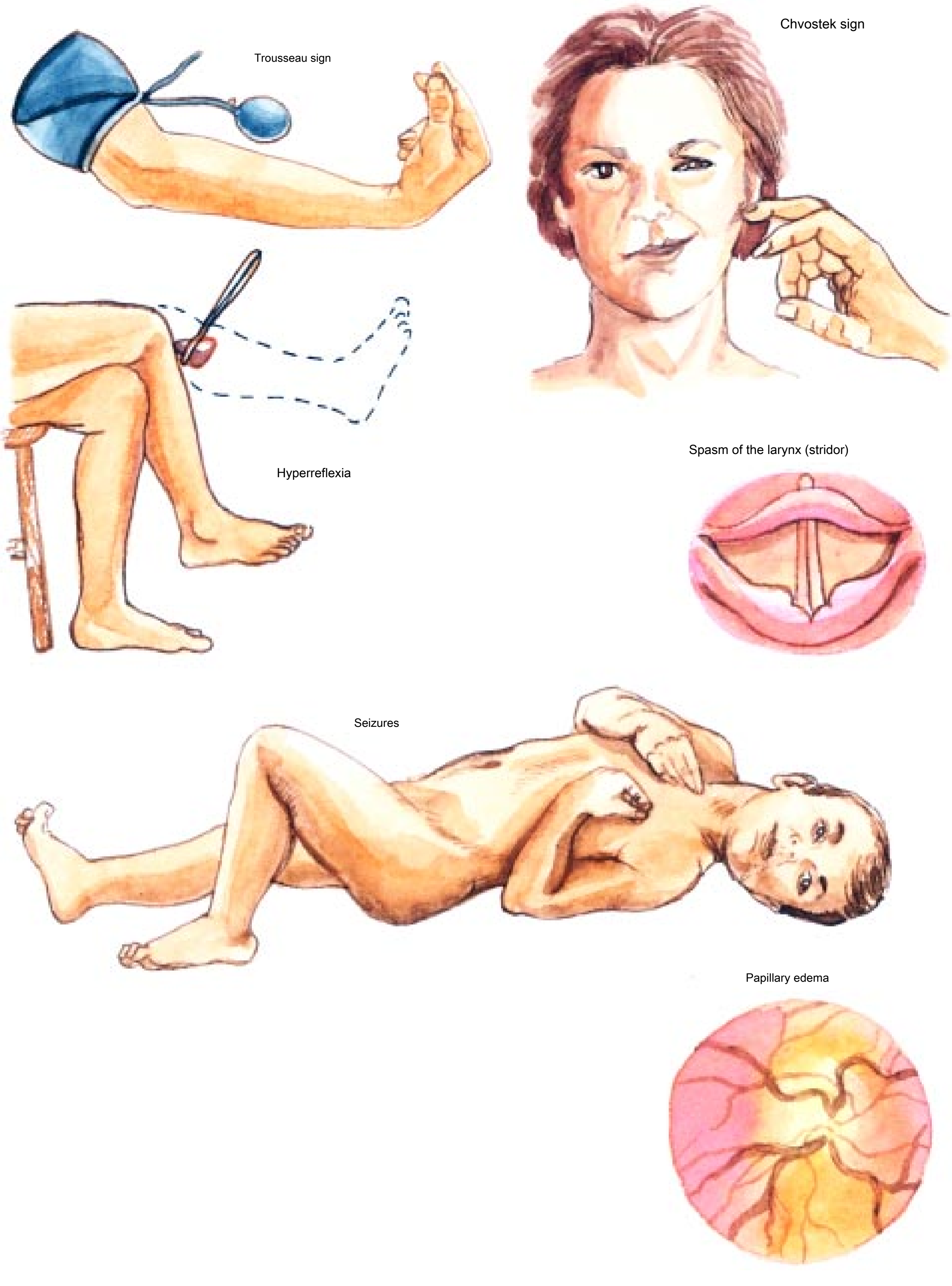

Trousseau sign
Chvostek sign
Hyperreflexia
Spasm of the larynx (stridor)
Seizures
Papillary edema

Fig. 74.8 Clinical manifestations of acute hypocalcemia.

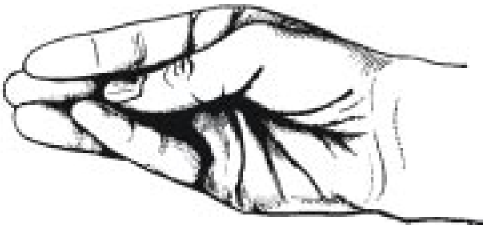


Fig. 74.9 Midwife's hand.

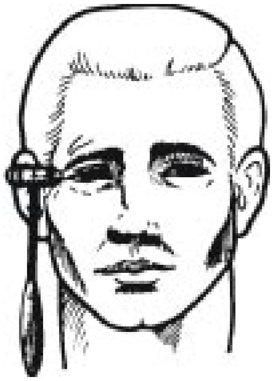


Fig. 74.10 Weiss sign.

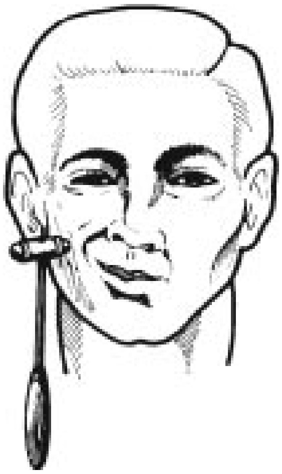


Fig. 74.11 Chvostek sign.

When forcefully pulling on an arm that is raised, contractures of the arm muscles are observed (Pool's sign), and consequently the midwife's hand is produced.

In tetany there is also an increase in direct muscle excitability. When the tongue is struck or percussed, a circumscribed depression (Schultze's sign) is formed.

Stimulation by cold or color of the nerve tract of a muscle causes local paresthesias or convulsions (Kashida sign).

Not only the motor nerves have hyperexcitability, as we have just seen, but also the sensory ones. Mechanical irritation of the trigeminal at its points

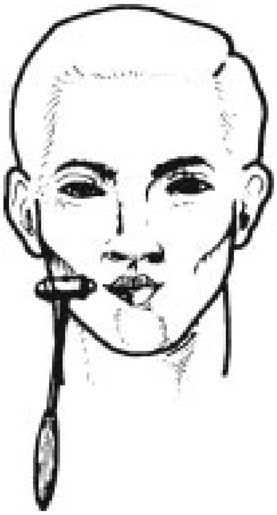


Fig. 74.12 Escherich sign.

Emergency causes local and immediate pain (Hoffmann's sign).

In hyperparathyroidism syndrome there is muscle hypotonia and none of the signs described are observed.

Provoked hyperpnea is an excellent biological test. The patient is laid down and encouraged to perform deep respiratory movements at a rate of 18/min; After 5-15 minutes, tetany is evident due to an acute attack.

Overt tetany or tetanic crisis

The stimuli for the production or triggering of a tetany crisis are usually simple: an emotion, an upset, etc.

Prodromes may be absent, but are sometimes manifested by dullness and tingling in the fingers, toes, and around the lips. Sometimes it is the existence of small fibrillar contractions in the muscles of the hands and feet or in the face. In severe tetany there are muscle cramps that affect isolated muscle groups of the hands and feet, in the form of carpopedal spasms.

From this moment on, the spasm generalizes to all the muscles of the skeleton and constitutes the typical attack:

1. In the upper extremities, the arms remain in approximation against the trunk (adduction). We can say that all muscle groups flex violently except the fingers, which in their three phalanges remain in extension and present a cone-shaped approach reminiscent of the midwife's hand.

2. In the lower extremities the tonic spasmodic contracture is in extension.

3. The muscle groups of the neck and spine suffer tonic contracture in hyperextension and the trunk adopts the opisthotonos position.
4. The so-called tetanic facies is very peculiar on the face due to the contraction of the facial muscle groups.
5. The eyeballs also suffer the contraction of their muscles, producing anisocoria, strabismus, and nystagmus.
6. The muscles of the trunk also contract: the abdominal walls, the intercostal muscles and the diaphragm contract on inspiration, contributing to cyanosis and even death from asphyxiation.
7. Laryngospasm, typical of children and infants, which can range from simple laryngeal stridor to apnea with cyanosis; sometimes it is fatal evolution.
8. The smooth muscles can also suffer, in which case the bronchus is affected (asthmatiform dyspnea), the esophagus (dysphagia), there may be vomiting, colic and rectal evacuation.

Patients retain consciousness (unlike epilepsy), although this acute and violent state can leave them absent from all conscious activity and there are even times when they are clouded.

Complementary exams

1. Electromyogram, which reveals neuromuscular hyperexcitability and shows repetitive potentials called doublets or triplets that are reproduced in bursts.
2. Electrocardiogram, showing QT prolongation.
3. Biochemical examinations:
   a) Very low calcium, less than 7 mg/dL and may reach 4 mg.
   b) Decreased calciuria, less than 100 mg in 24 hours.
   c) High phosphoremia above 6 mg/dL, which can reach 10 mg.
   d) Hypophosphaturia with decreased renal clearance of phosphorus.

   The association of frank hypocalcemia and hyperphosphoremia with a decrease in phosphocalciuria is totally in favor of parathyroid insufficiency with two very important reservations: preserved renal function and correction of the syndrome by the administration of parathyroid hormone.
4. Radiological examinations. Amputation of tooth roots is quite indicative; Sometimes there are hyperdensifications of the skeleton at the level of the cranial vault and the cortices of the long bones.

Etiology

In most cases, hypoparathyroid syndrome is iatrogenic0, as a consequence of the removal of the parathyroids during surgery due to thyroid or parathyroid disorders or due to reduced vascularization during hemostasis that causes ischemic necrosis.

Idiopathic is rare, few cases are known. It is discovered during childhood or adolescence.

ENDOCRINOMETABOLIC SYSTEM. ADRENAL GLANDS AND THEIR SYNDROMES

75

ANATOMY OF THE ADRENAL

The adrenal glands are two small endocrine organs of approximately 7-8 g in women and 8-9 g in men, which occupy the upper and inner part of the upper pole of both kidneys (Fig. 75.1l).

80% of it corresponds to the cortical portion. Its irrigation is extraordinary, the most abundant in the body. In normal condition it is yellow or yellowish brown.

The presence of accessory adrenal glands, whether complete or incomplete (in the latter eventuality, represented only by the cortical or medullary zone), is far from being a rarity and their possible existence must be kept in mind when considering the etiology of certain adrenal hyperfunctional syndromes, in which the exploration of the area where this gland is normally located is negative.

The accessory adrenal glands can be found in the vicinity of the normal ones, along the entire length of the abdominal aorta and its bifurcation, and in the vicinity of the gonads or inside them.

Each adrenal gland is made up of two completely different parts, according to its embryological origin, its anatomical structure and the functions it regulates: the cortical and the medullary. The clinical and humoral manifestations of the pathological processes following a functional alteration of these parts are also different, and determine a series of syndromes that can be perfectly individualized due to their well-defined characteristics. In this regard, it is convenient to remember that in many animal species they constitute two completely independent organs.

An exception to the general rule set out in the previous paragraph is adrenal tumor syndrome, formed by a set of symptoms and signs that accompany an increase in the size of the gland, since this increase offers similar characteristics to cortical or medullary tumors.

ADRENAL HISTOLOGY (FIG. 75.2)

The lipids of the adrenal cortex are: free cholesterol, cholesterol esters, triglycerides and phospholipids.

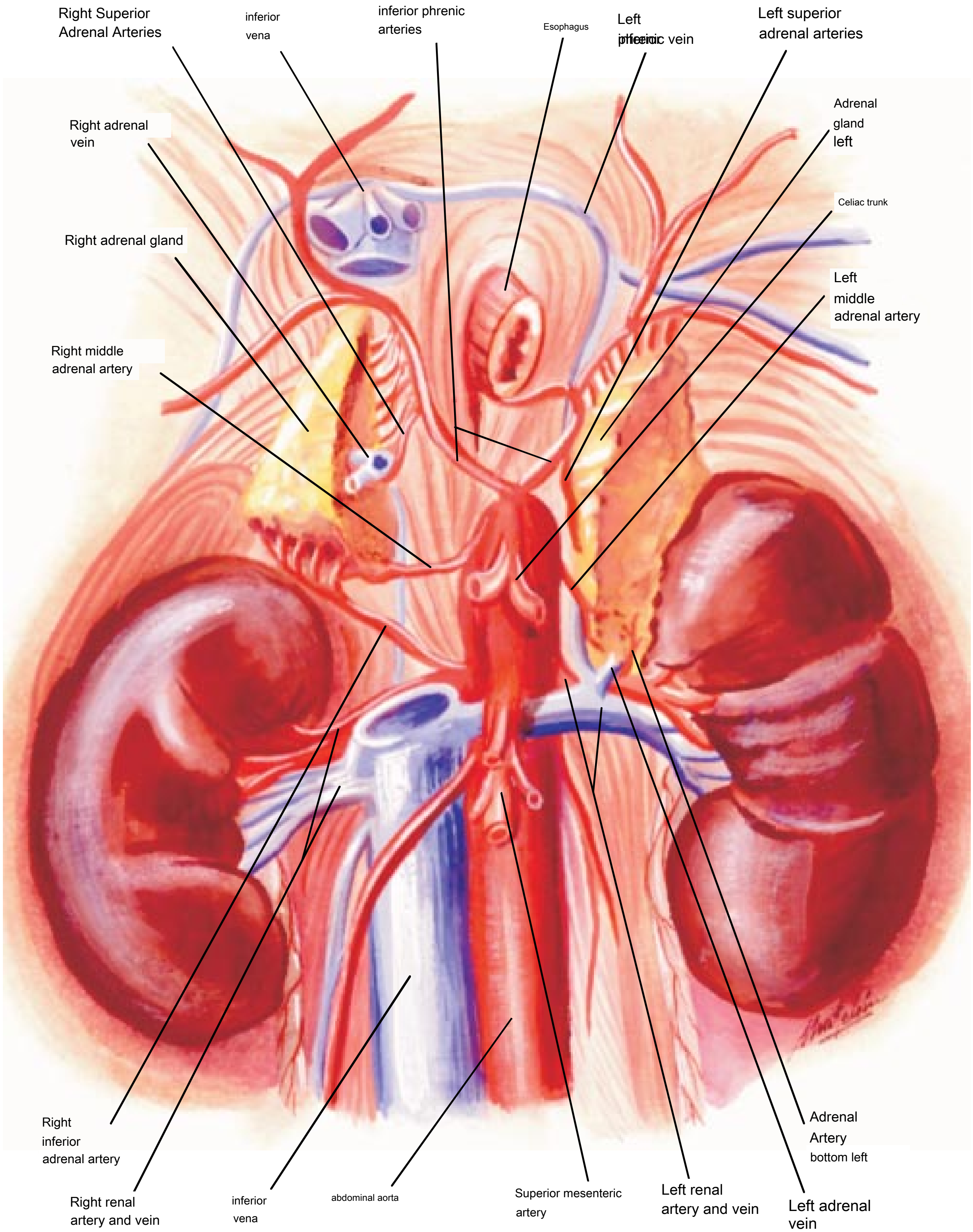
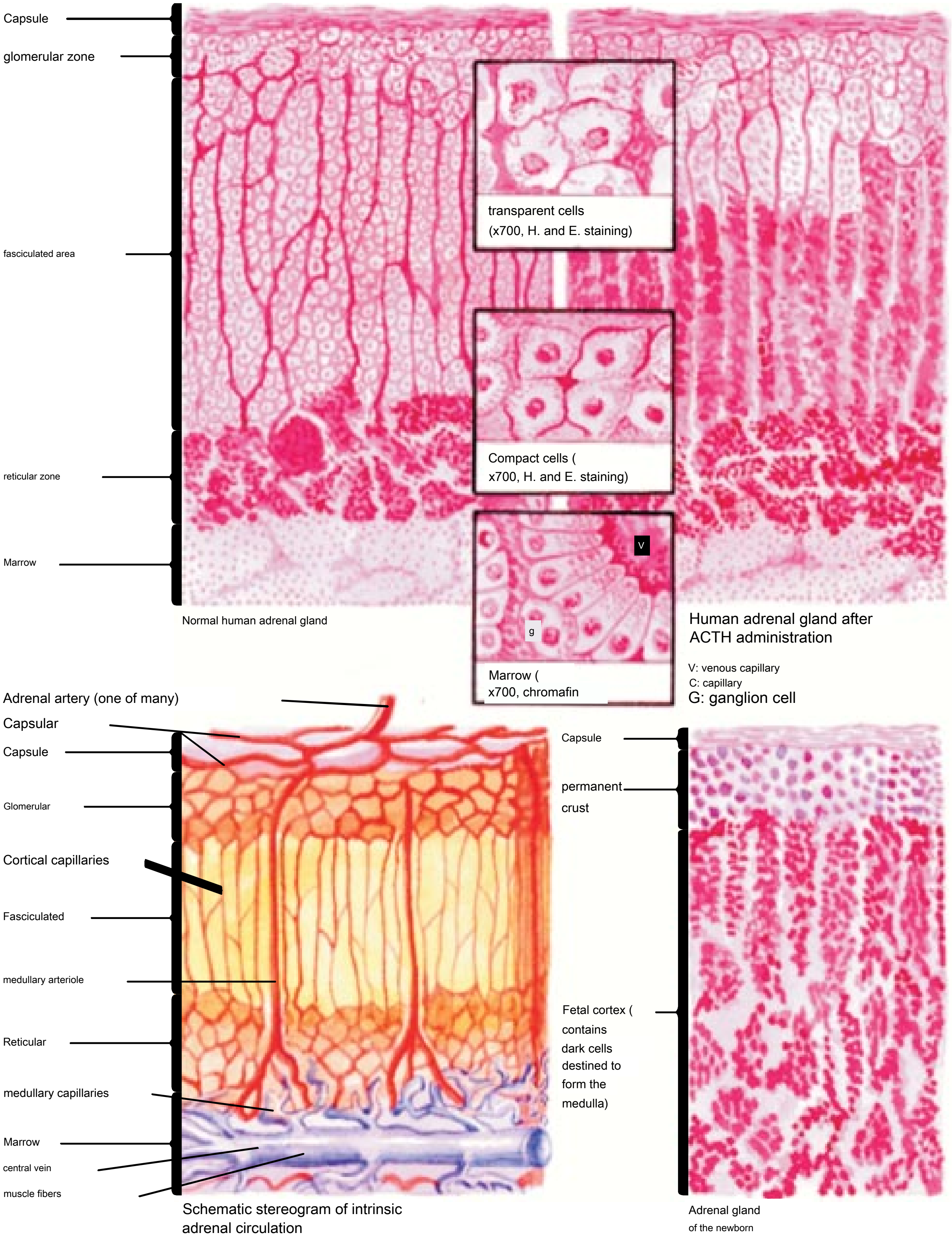

Right Superior Adrenal Arteries
inferior vena
inferior phrenic arteries
Esophagus
Left inferior phrenic vein
Left superior adrenal arteries
Right adrenal vein
Adrenal gland left
Celiac trunk
Right adrenal gland
Left middle adrenal artery
Right middle adrenal artery
Right inferior adrenal artery
Adrenal Artery bottom left
Right renal artery and vein
inferior vena
abdominal aorta
Superior mesenteric artery
Left renal artery and vein
Left adrenal vein

Fig. 75.1 Anatomy and blood supply of the adrenal glands.


Capsule
glomerular zone
fasciculated area
reticular zone
Marrow
transparent cells
(x700, H. and E. staining)
Compact cells (
x700, H. and E. staining)
V
g
Marrow (
x700, chromafin
V: venous capillary
C: capillary
G: ganglion cell
Adrenal artery (one of many)
Capsular
Capsule
Glomerular
Cortical capillaries
Fasciculated
medullary arteriole
Reticular
medullary capillaries
Marrow
central vein
muscle fibers
Capsule
permanent
crust
Fetal cortex (
contains
dark cells
destined to
form the
medulla)

Normal human adrenal gland

Human adrenal gland after ACTH administration

Schematic stereogram of intrinsic adrenal circulation

Adrenal gland of the newborn

Fig. 75.2 Histology of the adrenal glands.

In the normal gland there are very few preformed steroids, since when synthesized they quickly enter the circulation. The yellow coloring of the cortex depends on pigments (lipochromes) that are unrelated to glandular secretory activity.

Of the three net zones of the human adrenal cortex, the glomerular (external) zone, poorly developed, is made up of cells arranged in small groups and parallel to the fibrous capsule.

The zona fasciculata covers 80% of the normal gland. Cells are occupied by lipids, most of which are cholesterol, cholesterol esters and unsaturated lipids. These cells are called clear cells.

The zona reticularis is narrow and attached to the adrenal medulla. The cells in this area differ from clear cells, as they are rich in alkaline and acid phosphatases, ribonucleic acid and dehydrogenases. In their cytoplasm they contain abundant mitochondria. These cells have been called compact and constitute the functionally active cells of the cortex that supply the daily needs of corticosteroids.

The fasciculated zone is the storage area for precursor lipids, which are available for the synthesis of a greater amount of corticosteroids, when the gland is stimulated. In long-lasting or chronic alarm states, or after stimulation with exogenous ACTH (exogenous corticotropin), clear cells become compact cells, to the extent that the lipids they store are converted into steroid hormones.

The adrenal medulla comes from the primitive cells of the neural crest, which migrate to the center of the fetal adrenal and form the medulla, with its complement of mature cells, the pheochromocytes.

In the fetus, accumulations of cells are seen along the abdominal aorta. Other groups are dispersed throughout the sympathetic nervous system in relation to the cervical and thoracic ganglia, gastrointestinal tract, gallbladder, heart and gonads. After birth, these islets are replaced by lymphatic tissue, but they can persist and cause extra-adrenal pheochromocytomas.

PHYSIOLOGY OF THE ADRENAL

The adrenal cortex secretes a group of hormones called corticosteroids and also androgenic hormones. The adrenal medulla secretes the hormones adrenaline and norepinephrine, in response to sympathetic stimulation.

ADRENAL CORTEX

The corticosteroids it produces are of two main types: mineralocorticoids and glucocorticoids. In addition, it produces small amounts of androgens whose effects are similar to those of the male sex hormone: testosterone.

The name mineralocorticoids is due to the fact that these hormones act mainly on the electrolytes of extracellular fluids: sodium, potassium and chlorides.

Glucocorticoids were so called because one of their main effects is to raise the concentration of glucose in the blood.

Function of mineralocorticoids

Mineralocorticoids, including aldosterone, are produced in the glomerular zone.

When adrenal corticosteroid secretion is suppressed, death occurs. In the absence of mineralocorticoids, the concentration of sodium and chlorides in the extracellular fluids decreases considerably, as does the volume of the extracellular fluid; Cardiac output decreases and a shock-like state is established, followed by death. These events are prevented by administering aldosterone or another mineralocorticoid and salt.

Mineralocorticoids are basically represented by aldosterone, which is responsible for 95% of this activity, to which corticosterone contributes, which also has glucocorticoid effects, and deoxycorticosterone, which is secreted in small quantities, whose effects are equal to aldosterone, but 30 times less potent.

In summary, the fundamental effects of mineralocorticoids are:

- Increased sodium reabsorption by the renal tubule.

Increase in renal potassium excretion.

These fundamental effects were exposed when studying the hydromineral balance (Chapter 68) (see figs. 68.5 and 68.6) and will be treated again in the explanation of the syndromes.

Role of glucocorticoids (Fig. 75.3)

Glucocorticoids are mainly produced in the fascicular zone.

The adrenalectomized animal can survive with the administration of mineralocorticoids, but it is not a normal animal. His metabolic system to use carbohydrates, proteins and fats is altered, which is why he cannot resist physical or psychological attacks or minor illnesses, which can cause death.

Therefore, glucocorticoids have functions as important for the life of the animal as mineralocorticoids.

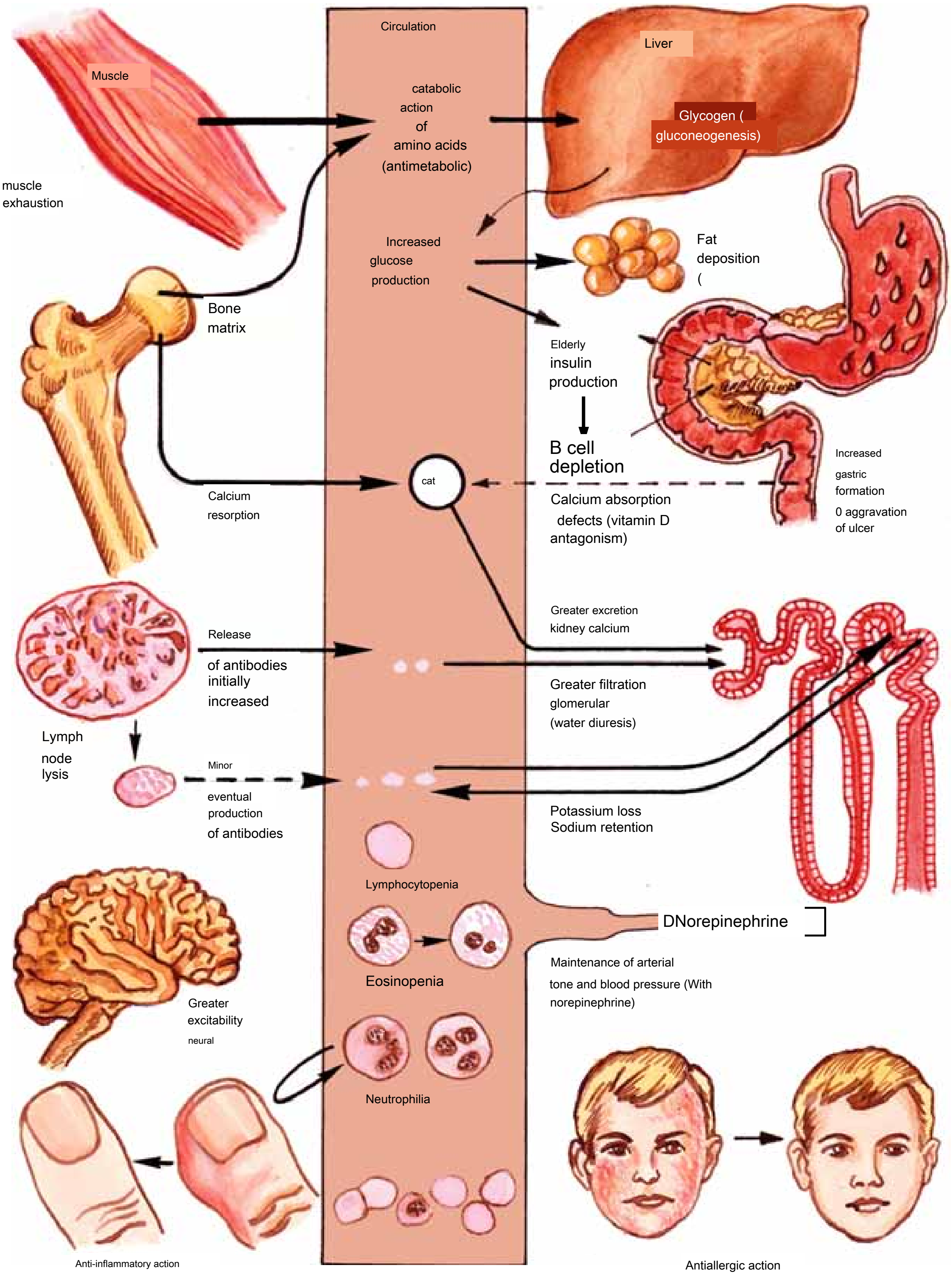

Circulation
Liver
Muscle
catabolic action of amino acids (antimetabolic)
Glycogen (gluconeogenesis)
muscle exhaustion
Increased glucose production
Fat deposition (
Bone matrix
Elderly insulin production
B cell depletion
cat
Calcium resorption
Calcium absorption defects (vitamin D antagonism)
Increased gastric formation
0 aggravation of ulcer
Greater excretion kidney calcium
Release of antibodies initially increased
Greater filtration glomerular (water diuresis)
Lymph node lysis
Minor eventual production of antibodies
Potassium loss
Sodium retention
Lymphocytopenia
DNorepinephrine
Eosinopenia
Maintenance of arterial tone and blood pressure (With norepinephrine)
Greater excitability neural
Neutrophilia
Anti-inflammatory action
Antiallergic action

Fig. 75.3 Biological actions of glucocorticoids (cortisol).

95% of this activity is due to cortisol (also called hydrocortisone and compound F), to a lesser extent to corticosterone and to a much lesser extent to cortisone.

The best known effect of cortisol and other glucocorticoids on metabolism is their ability to increase hepatic gluconeogenesis, favoring an increase in hepatic glycogen, a decrease in the use of glucose by cells and an increase in blood glucose concentration, which causes adrenal diabetes.

In addition, there are important effects on protein and fat metabolism and also hematological effects on inflammatory processes, immunity, etc.

sex hormones

They are produced in the reticular zone, mainly androgens (C19) and small amounts of estrogenic hormones (C18), both in quantities that have no biological importance, compared to the production of the gonads.

ADRENAL CORD

The cells of the adrenal medulla are of ectodermal origin and come from the neural crest. The adrenal medulla is essentially a sympathetic ganglion, in which the postganglionic cells have lost their axons and have specialized in secreting their products directly into the bloodstream.

Sensory stimuli traveling through the brain stem or messages ascending through the spinal cord converge in the hypothalamus, from where they are conducted downwards again through the spinal cord. The sympathetic processes from T10 to LI form the splanchnic nerves, which, as preganglionic fibers, establish synapses in the preaortic ganglia and continue towards the adrenal medulla. There, the polyhedral chromaffin cells are stimulated by acetylcholine released in the nerve terminals and secrete epinephrine and norepinephrine. It appears that cells containing acid phosphate preferentially secrete epinephrine, while those showing fluorescence secrete norepinephrine. The adrenal medulla of the fetus contains only norepinephrine, epinephrine appears some time after birth. Chromaffin cells in the spinal cord respond to stimulation of the splanchnic nerve during emergency situations. Insulin hypoglycemia will deplete the cells that secrete epinephrine specifically, while reserpine, in small doses, causes the loss of norepinephrine exclusively.

The biosynthesis of catecholamines is carried out by the hydroxylation and decarboxylation of phenylalanine and tyrosine is formed. This is converted into dihydroxyphenylanine (DOPA) by the enzyme tyrosinase and is then transformed into dopamine (hydroxytyramine), which in turn, through an a-hydroxylase, is converted into norepinephrine, which undergoes N-methylation and gives rise to epinephrine.

The enzyme responsible for converting norepinephrine into epinephrine is found in high concentrations only in the adrenal medulla. The average content of the combined norepinephrine-epinephrine of the normal human adrenal medulla ranges from 2-4 mg/g tissue in the adult, of which only up to 30% is norepinephrine, while in all extramedullary tissues that secrete catecholamines, the majority of this catecholamine is norepinephrine. Both epinephrine and norepinephrine differ considerably in their pharmacophysiology.

At one end of the scale, norepinephrine has a much greater effect on blood pressure than epinephrine. The first acts by producing vasoconstriction in most of the arteriolar territory in the body; The second, although it has a similar action on the arterioles of the skin (which leads to paleness), dilates the blood vessels in the liver and muscle.

Norepinephrine, while raising blood pressure through vasoconstriction, tends to decrease the cardiac index; in contrast, epinephrine increases it. Epinephrine also increases basal metabolism, while norepinephrine does so only slightly. Epinephrine causes marked bronchial dilation and inhibition of intestinal motility, but norepinephrine does not. Epinephrine, by activating glycogenolytic enzymes and inhibiting insulin secretion, leads to a marked hyperglycemia not also shared by norepinephrine. It also increases the irritability of the central nervous system, causing a feeling of imminent anxiety and a marked dilation of the pupils. Adipose tissue lipase is activated by both catecholamines, leading to lipolysis and an increased discharge of non-esterified fatty acids into the bloodstream, which rise above normal (0.4 ± 0. 2 mEq/L). It is interesting to know that patients affected by tumors that produce excess catecholamines are almost never fat. Epinephrine, much more than norepinephrine, produces neutrophilia with eosinopenia. All of these activities favor the immediate survival of an animal taking part in a fight, during flight, or while possessed by fear. This was first pointed out by Cannon in his theory of the alarm reaction and later extended by Selye to the general adaptation syndrome, which also includes a secondarily increased adrenal cortico-adrenal activity.

The metabolism of catecholamines takes place mainly in the liver; It consists of orthomethylation (80%) and oxidative deamination (20%) by a catechol-0-methyltransferase (COMT) and a monoamine oxidase (MAO). Less than 4% of intravenously administered epinephrine or norepinephrine is found in urine; However, considerable amounts of metanephrine, normetanephrine, and vanilmandelic acid are normally found and can serve for the quantitative determination of marrow activity.

REGULATION OF ADRENAL SECRETION OF STEROIDS

The inner part of the cortex (fascicular and reticular zones) is under the control of ACTH and secretes all adrenal steroids except aldosterone.

The outer part of the cortex (zona glomerulare) secretes aldosterone in response to the stimulus of angiotensin II, potassium, and

ACTH activates adrenal adenyl cyclase, stimulating the formation of AMPe. This initiates the formation of a protein that, directly or indirectly, catalyzes the conversion of cholesterol to 20-a-hydroxycholesterol, which in turn is converted to pregnenolone, which serves as a precursor to all adrenal steroids.

Under normal conditions, cortisol and dehydroisoandrosterone are secreted more abundantly, but the deficiency of one of the enzymes involved in the conversion of pregnenolone to its normal derivatives produces an increase in the intermediates formed before the disturbed stage.

This distortion is observed in congenital adrenal hyperplasia, some adrenal neoplasias, and in treatment with pharmacological inhibitors of adrenal enzymes.

The secretory activity of the adrenal cortex is under direct control of ACTH, which is why problems related to the regulation of adrenal steroid secretion are transformed into problems related to the controls of ACTH secretion, which are due to the following factors:

The first is the concentration of cortisol. Of all the products secreted by the human adrenal, cortisol is the only one with strong potency to regulate ACTH. The higher the cortisol concentration, the greater the inhibitory action on ACTH secretion: the lower the cortisol level, the less the inhibition of ACTH secretion, which translates into an increase in ACTH secretion.

The second is the sleep regimen. In normal people who sleep at night, plasma ACTH and cortisol secretion increase from two in the morning with a maximum when waking up; Then it decreases and its values go down throughout the day, until reaching the minimum at night.

The third factor is the alarm call in the face of aggressive situations: hypoglycemia, major surgery, pyrogens, etc., which increase the secretion of ACTH.

In practice, plasma cortisol concentration is used as an indirect index of ACTH secretion. Within physiological limits of ACTH concentration, the adrenal response is proportional to the amount of ACTH that reaches the gland and the secretion of cortisol occurs, which is maintained as long as the plasma concentration of ACTH persists. When this decreases, the plasma concentration of cortisol falls, which undergoes a half-disintegration in 1-2 hours.

Aldosterone secretion, on the other hand, is only partially under the control of ACTH and is fundamentally influenced by sodium losses or any cause that produces hypovolemia. In these cases, renin production occurs in the kidneys, which catalyzes angiotensin I, transforming it into angiotensin II, which stimulates the production of aldosterone (see also, Chapter 68).

This renin-angiotensin system is the main regulator of aldosterone secretion, at least in most of the processes we know as secondary aldosteronism.

STEROID BIOSYNTHESIS

All steroid hormones basically have a similar structure with minor chemical differences, which give rise to a notable specificity in their biological action. They also have a basic skeleton of 17 carbon atoms marked in a well-defined order, to which 1, 2 or 4 carbon atoms are added.

Figure 75.4 presents in simplified form the complex biosynthesis of corticosteroids from cholesterol and possibly acetate, stimulated by ACTH.

ADRENAL SYNDROMES

Multiple classifications are possible (excluding iatrogenic hypercorticisms).

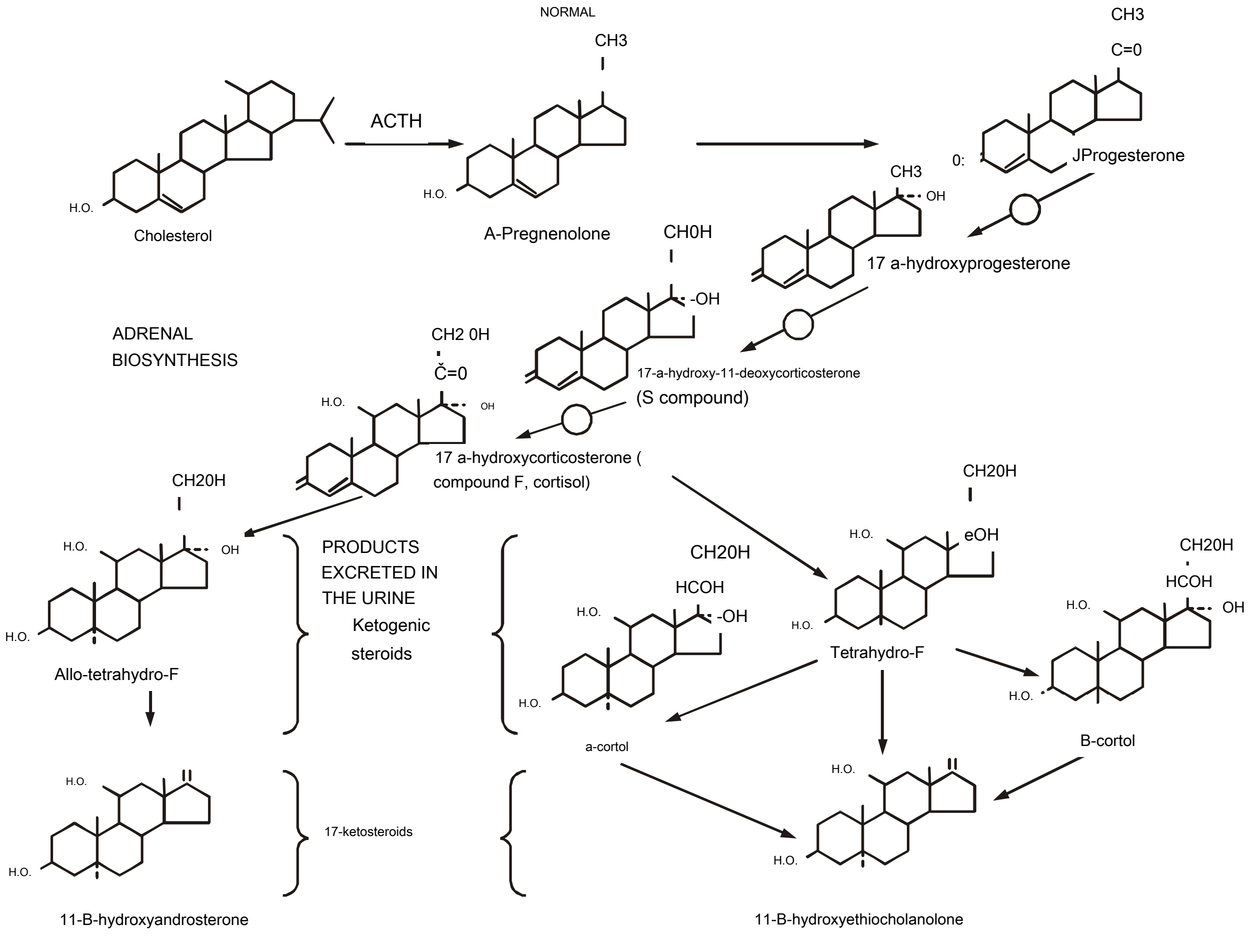

NORMAL
CH3
CH3
C=0
ACTH
H.O.
Cholesterol
H.O.
A-Pregnenolone
0:
JProgesterone
CH3
OH
CH0H
17 a-hydroxyprogesterone
-OH
ADRENAL
BIOSYNTHESIS
CH2 0H
Č=0
17-a-hydroxy-11-deoxycorticosterone
(S compound)
H.O.
OH
17 a-hydroxycorticosterone (compound F, cortisol)
CH20H
CH20H
H.O.
OH
H.O.
Allo-tetrahydro-F
PRODUCTS EXCRETED IN THE URINE
Ketogenic steroids
CH20H
HCOH
-OH
H.O.
H.O.
a-cortol
H.O.
eOH
H.O.
Tetrahydro-F
CH20H
HCOH
OH
H.O.
H.O.
B-cortol
H.O.
H.O.
11-B-hydroxyandrosterone
17-ketosteroids
H.O.
H.O.
11-B-hydroxyethiocholanolone

Fig. 75.4 Chemical formulas of adrenal steroid biosynthesis.

We will use the following:

I. Cortical syndromes.

- A. Adrenal corticohyperfunction.
  - 1. Mmetabolic hypercorticisms:
    - a) Pure metabolic hypercorticism: Cushing's syndrome.
    - b) Metabolic hypercorticisms accompanied by hypersecretion of mineralocorticoids or androgens.
  - 2. Androgenic hypercorticisms:
    - a) Pure androgenic hypercorticism: adrenogenital or Cooke-Apert-Gallais
    - b) Androgenic hypercorticism accompanied by metabolic, estrogenic or mineral hypercorticism.
  - 3. Estrogenic hypercorticism: syndrome due to excess estrogen (feminization).
  - 4. Hypermineralocorticisms:
    - a) Primary hyperaldosteronism: syndrome of Conn.
    - b) Secondary hyperaldosteronism.
- B. Adrenocortical hypofunction.
  - 1. Acute adrenal corticosteroid insufficiency: Waterhouse-Friderischsen syndrome.
  - 2. Chronic adrenal corticosteroid insufficiency: Addison's disease.

II. Spinal syndrome.

- A. Bone marrow hyperfunction (pheochromocytoma).

III. Tumor syndrome.

Of the adrenal syndromes in this classification, we will only describe a few.

PURE METABOLIC HYPERCORTICISM: CUSHING SYNDROME

Concept

Cushing's syndrome is characterized by cortical hyperfunctioning with increased glucocorticoids, which dominate the clinical picture; However, it is frequently associated with a increase in mineralocorticoids and sometimes, with symptoms determined by androgen hypersecretion.

Syndromogenesis or pathophysiology (see Fig. 75.3)

Excess cortisol has an effect on three essential metabolisms:

1. Carbohydrate metabolism. The hyperglycemic action of cortisol is exerted, perhaps partially, by inhibition of the peripheral use of glucose by opposing phosphorylation in muscle and adipose tissue: but it reflects, above all, the increase in hepatic neoglycogenesis, by activating cortisol, the enzymes that intervene in it, such as:

   pyruvatecarboxylase, fructose 1-6 diphosphatase and glucose 6-phosphatase. The diabetes thus induced will usually be reversible after suppression of hypercortisolism; In some cases it may persist, especially in genetically predisposed subjects.
2. Metabolism of proteins. Cortisol favors protein catabolism, makes the nitrogen balance negative and reduces the incorporation of amino acids into the muscles. Protein catabolism plays a major role in the growth interruption observed in the child, independently of the antagonistic action of cortisone with respect to the somatotropic hormone.

3. Lipid metabolism. Cortisol favors the synthesis of long-chain fatty acids, ketogenesis and ketosis, probably by increasing insulin release. This lipotropic action gives rise to a deposit of *"dark" fat in particular points: face, trunk, abdomen and, in the form of a ball of fat, at the base of the neck.

Electrolyte balance. Hydrocortisone promotes sodium retention and potassium elimination. This last action, the most apparent, leads to hypokalemia that requires considerable amounts of potassium to be corrected, due to the decrease in intracellular potassium storage.

Hematopoiesis. Hematological changes are the result of a double action of the ACTHy of corticosteroids: lytic action on lymphocytes and eosinophils and bone marrow stimulation action, which affects neutrophil granulocytes and erythroblasts.

Some manifestations of Cushing's syndrome are not so easily explained. An example is high blood pressure. The role of adrenal corticosteroid hormones is not in doubt, but their relative responsibility remains poorly understood; has not been demonstrated an excess of mineral hormones by determinations; The role of increased blood mass, in turn linked to sodium retention, remains hypothetical, as does the intervention of androgens, to whose atherogenic effect hypertension has been attributed.

Amenorrhea is not entirely attributed to hyperandrogenia. It is reproducible by cortisone in large doses: a hypothalamic-pituitary link can explain the cases in which it is associated with ovarian hypoplasia. Sometimes pituitary radiotherapy causes a recurrence of menstruation; However, the level of gonadotropins is generally normal.

As for psychological disorders, they depend in part on excess cortisol, since they follow the fluctuations of the disease. However, hypercorticism is often limited to revealing a pre-established psychotic state which, in turn, may be the cause of the glandular disturbance;

The parallel with Basedow's disease is striking.

Syndromography or positive diagnosis

Clinical picture (fig. 75.5)

1. Symptoms that show increased glucocorticoids:

   a) Obesity. Sometimes considerable and quick to install. It is characterized, above all, by its faciotroncular distribution, which infiltrates the face, the nape of the neck (giving the buffalo neck) and the trunk. It contrasts with the thinness of the limbs, specifically in their distal part, accentuated by muscle atrophy. It is accompanied by changes in the skin, which becomes thin, dry, erythematous, with violet or purplish red vergetures or stretch marks, located on the flanks, buttocks and thighs.

   b) Generalized osteoporosis.

   c) Symptoms of diabetes: polyuria, polydipsia, etc.

2. Symptoms that show the increase in mineralocorticoids:

   a) Permanent arterial hypertension, both maximum and minimum.

   b) Signs of arteriolar suffering: capillary fragility with ecchymosis and purpura. Positive loop sign.

   c) Chronic glomerulonephritis, sometimes.

3. Symptoms that show increased androgens.

   The anabolic activity of androgens is masked by the catabolic activity of glucocorticoids.

   Sexual modifications are less intense than in adrenogenital syndrome and are not constant. It is especially noticeable in women: development of hirsutism and menstrual disorders.

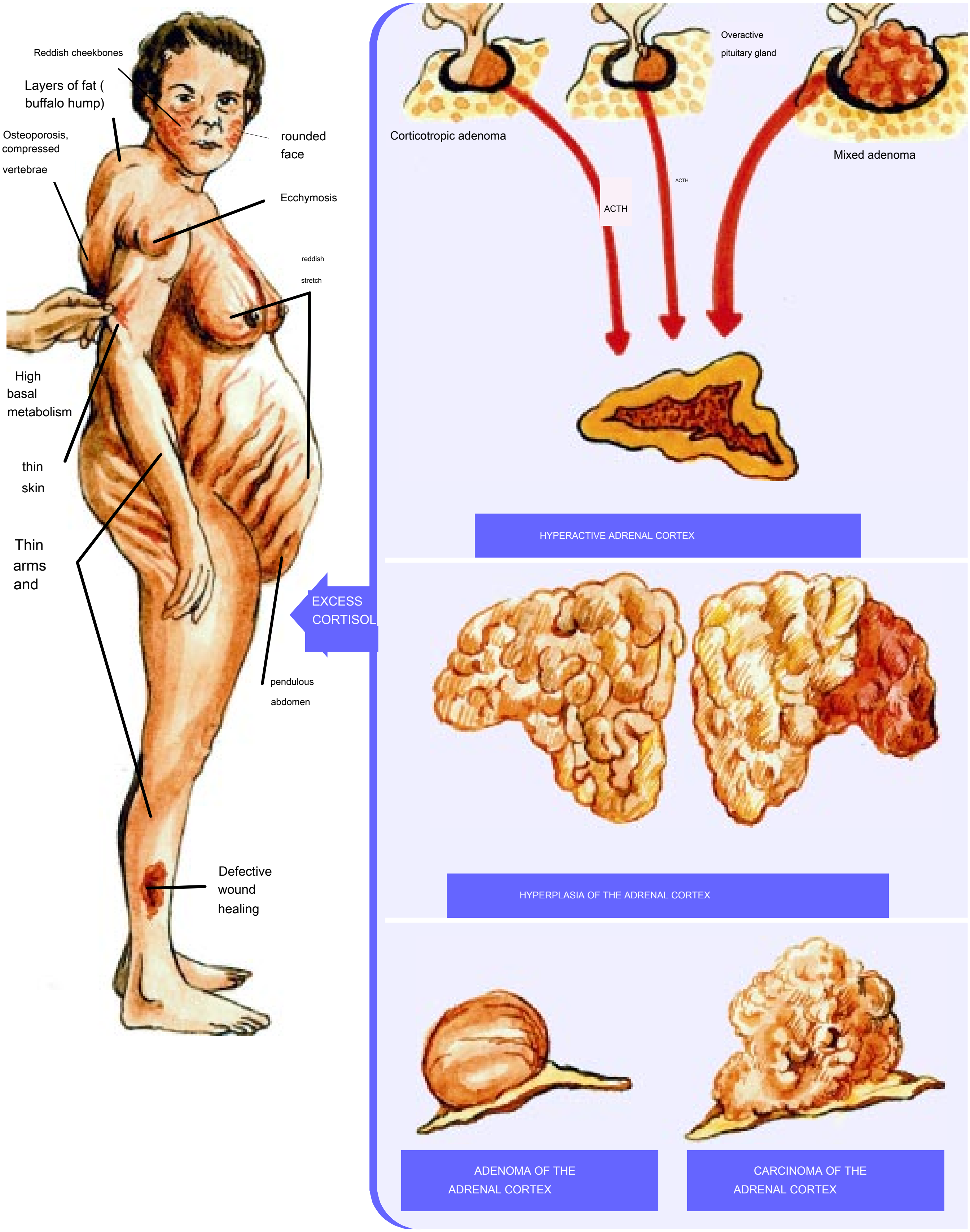

Reddish cheekbones
Layers of fat (buffalo hump)
Osteoporosis, compressed vertebrae
rounded face
Ecchymosis
reddish stretch
High basal metabolism
thin skin
Thin arms and
pendulous abdomen
Defective wound healing
EXCESS CORTISOL
Overactive pituitary gland
Corticotropic adenoma
Mixed adenoma
ACTH
ACTH
HYPERACTIVE ADRENAL CORTEX
HYPERPLASIA OF THE ADRENAL CORTEX
ADENOMA OF THE ADRENAL CORTEX
CARCINOMA OF THE ADRENAL CORTEX

Fig. 75.5 Cushing syndrome.

Complementary exams

1. That show the increase in glucocorticoids:
   a) Blood count: polyglobulia, neutrophilia with lymphocytopenia and eosinopenia.
   b) Hyperglycemia: almost constant.
   c) Free cortisol in 24-h urine and elevated plasma cortisol.
   d) Elevation of urinary 17-hydroxycorticoids and 17-ketosteroids.
   e) Inhibition test with 2 and 8 mg dexamethasone.
   f) Determination of ACTH, which is elevated if it is of pituitary cause and decreased if the hyperfunction is of adrenal cause.
2. That show the increase in mineralocorticoids:
   a) Hypernatremia.
   b) Hypokalemia.
3. That show the increase in supra-androgens kidneys:
   a) Increase in urinary excretion of 17-ketosteroids.
   b) Increase in plasma androstenedione and dehydroepiandrostenedione.

Etiology (see Fig. 75.5)

The original description by Harvey Cushing in 1932 was the presence of a basophilic cell adenoma in the pituitary gland; For years the disease was considered primarily pituitary and with secondary effects on the adrenals.

Today we consider three main groups:

1. Autonomous production of cortisol due to a primary lesion (benign or malignant) of the adrenal cortex: hyperplasia (Fig. 75.6), adenoma (single or multiple) and functioning carcinoma.
2. Excessive secretion of ACTH by the pituitary gland that stimulates the secretion of cortisol (corticotropic tumor or basophilic hyperplasia) (fig. 75.7): or a functional alteration at the level of the hypothalamus. The term basophilic tumor has fallen into disuse and has been replaced by corticotropic tumor.
3. Ectopic production of ACTH by non-pituitary neoplasms (lung carcinoma, thymomas, medullary thyroid carcinoma, insular pancreatic cell tumor, etc.).

Therapeutic administration, over a period of time, of cortisol or similar synthetic steroids produces the same clinical picture (iatrogenic Cushing's syndrome).

The name Cushing's disease is reserved for cases similar to those described by the author, and Cushing's syndrome, for the clinical and metabolic disorder of the other causes.

When the hyperfunction of the adrenals is due to a pituitary lesion, we will then have Cushing's disease and, as we already said, this lesion can be a corticotropic adenoma of the pituitary or a simple pituitary basophilism (hyperplasia).

But whatever its etiology, increased cortisol is responsible for most of the symptoms (see physiology).

PURE ANDROGENIC HYPERCORTICISM: ADRENOGENITAL OR COOKE-APERT-GALLAIS SYNDROME

Concept

Adrenogenital syndromes are the set of clinical conditions characterized by more or less accentuated changes in the patient' s sexual sphere: anatomical (genital and extragenital), functional and psychological, due to androgen hyperfunction by the adrenal cortex.

Syndromogenesis and syndromography

There are two fundamental enzymatic disorders: 21-hydroxylase deficiency (the most common cause of congenital adrenal hyperplasia) and 11-B-hydroxylase deficiency (second in order of frequency). In both cases, the normal biosynthesis of cortisol is blocked and when inhibition is lost due to negative feedback, ACTH is produced in excess and adrenal hyperplasia occurs. The same could be explained in older children or in adults, in whom the enzymatic defect, which is less serious, is diagnosed later.

21-hydroxylase deficiency

Because of this deficit, the synthesis of cortisol is somewhat disturbed at the stage in which 17-hydroxyprogesterone must be converted to 11-deoxycortisol and the synthesis of aldosterone is disturbed at the stage in which progesterone is converted to 11-deoxycorticosterone.

The hypersecretion of 17-hydroxyprogesterone causes the urinary excretion of a specific metabolite for the diagnosis, pregnantriol.

Adrenal androgens do not require 21-hydroxylation for their synthesis and are produced in large quantities, with high urinary values of 17-ketosteroids. The clinical consequence is virilization, which depends on sex, the age at which it occurs and the intensity of the process.

The female fetus is very sensitive to androgens and in these cases of virilizing congenital adrenal hyperplasia, partial masculinization of the genitourinary system occurs, in those parts that are the last to differentiate.

Symptoms

Unless the boy or girl dies or is treated, the patient grows rapidly and has strong muscle development.

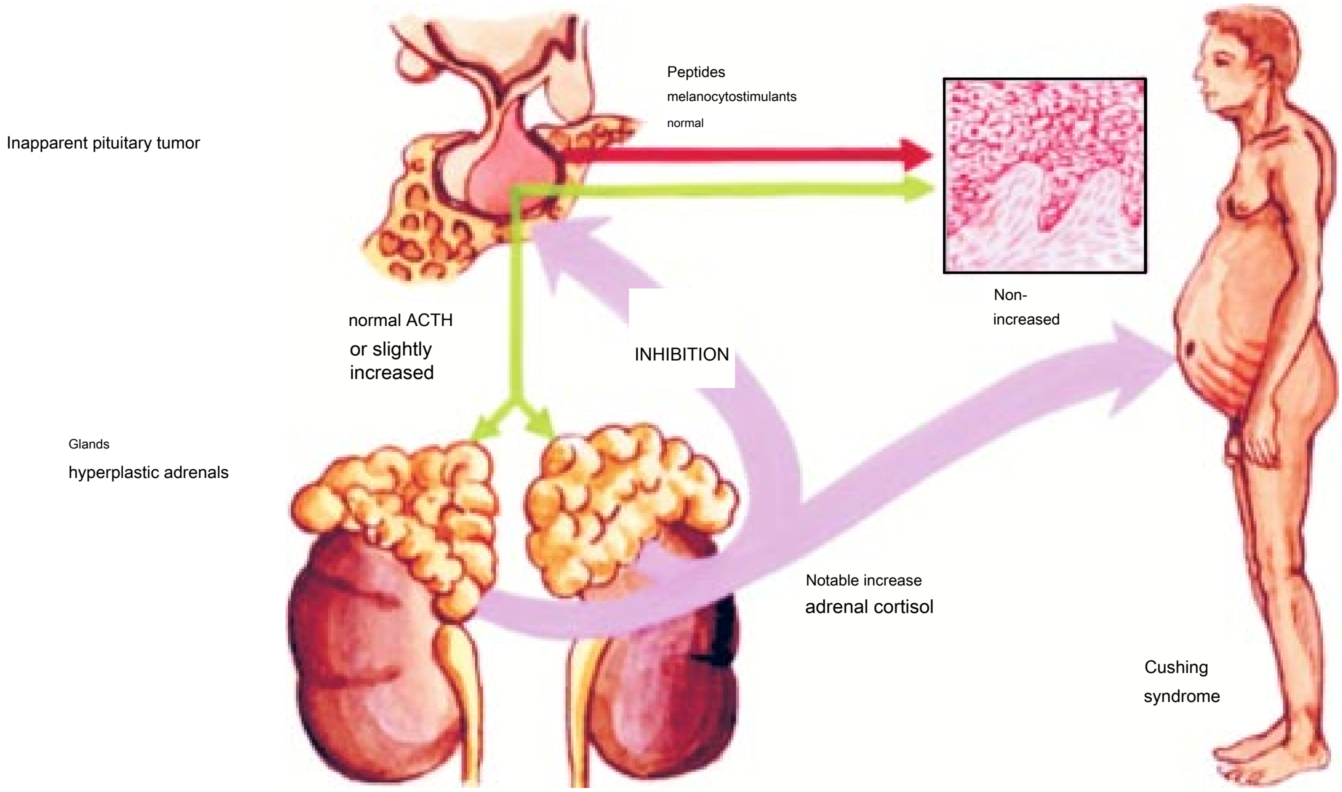
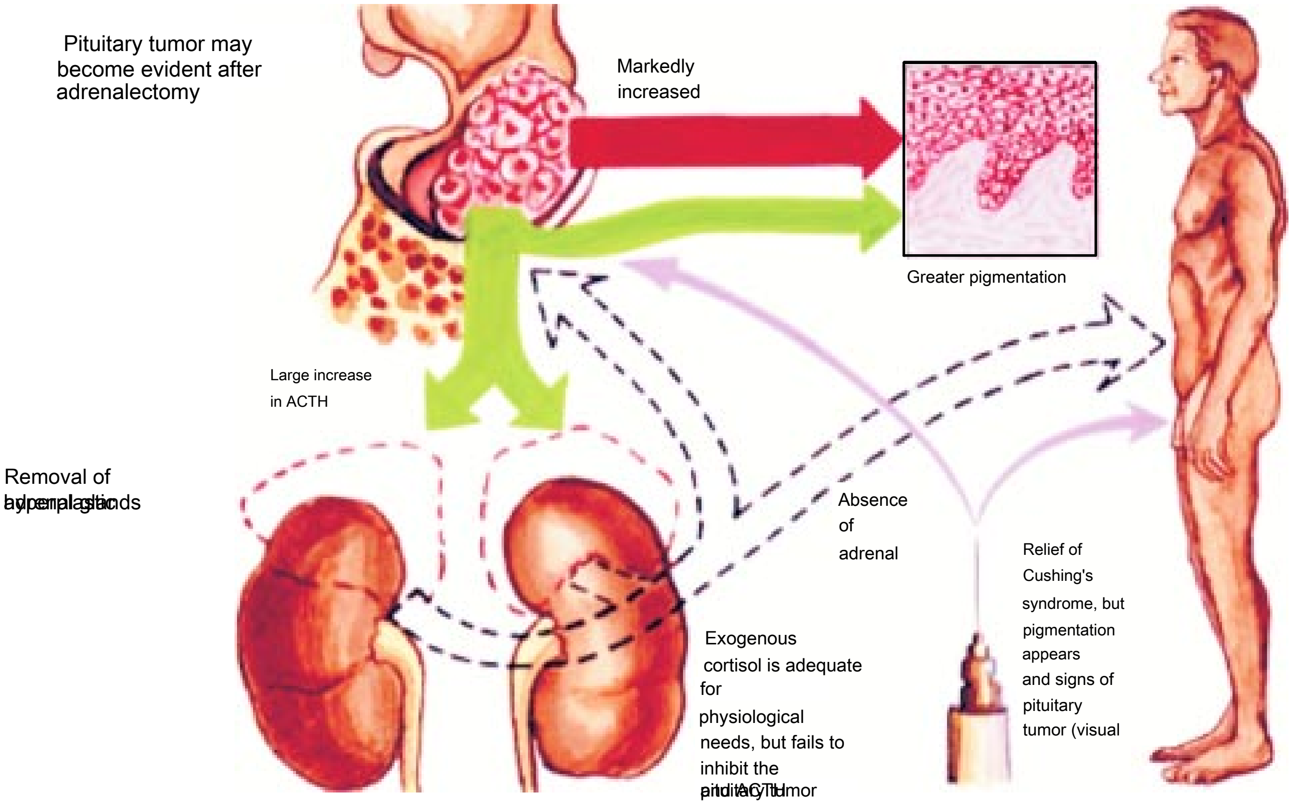

Inapparent pituitary tumor
Peptides
melanocytostimulants
normal
normal ACTH
or slightly
increased
INHIBITION
Non-
increased
Glands
hyperplastic adrenals
Notable increase
adrenal cortisol
Cushing
syndrome

Fig. 75.6 Hyperplasia of the adrenal glands.

Fig. 75.7 Corticotropic tumor of the pituitary gland.


Pituitary tumor may
become evident after
adrenalectomy
Markedly
increased
Greater pigmentation
Large increase
in ACTH
Removal of
adrenal glands
hyperplastic
Absence
of
adrenal
Relief of
Cushing's
syndrome, but
pigmentation
appears
and signs of
pituitary
tumor (visual
Exogenous
cortisol is adequate
for
physiological
needs, but fails to
inhibit the
pituitary tumor
and ACTH

Between 2 and 10 years of age, pubic and axillary hair appears, followed by facial hirsutism and deep voice tone. Bone age and height are advanced in relation to real age. Premature closure of the epiphyses predestines the patient to a smaller height in adult life. Except for the symptoms of precocious puberty, the boy may appear normal. The untreated female shows progressive virilization: she lacks female sexual characteristics, she has no mammary or uterine development, nor menstrual cycles at puberty.

Complementary exams

1. High concentration of pregnantriol in the urine.
2. Increased 17-ketosteroids.
3. Normal 17-hydroxycorticoids.
4. Easy suppression of alterations with the administration of glucocorticoids.

11-B-hydroxylase deficiency

It is much less frequent. The final stage of cortisol synthesis is disturbed, cortisol secretion is decreased, ACTH secretion is excessive, and large amounts of 11-deoxycortisol are eliminated. When the synthesis of corticosterone is disturbed, 11-deoxycorticosterone, a mineralocorticoid that causes arterial hypertension, is secreted in large quantities.

Adrenal androgens are produced in excessive amounts in response to excess ACTH. Virilization occurs whose pathophysiology is similar to the previous one.

Symptoms

The most obvious clinical manifestations are hypertension and virilism.

Complementary exams

1. Increased excretion of 11-deoxycortisol in urine.
2. Low plasma levels of renin and aldosterone.
3. Increase in 17-hydroxycorticoids in plasma and urine.

Adrenal tumors

They produce a clinically manifest excess of androgens and cortisol. Tumors that secrete only androgens are less common.

They are malignant, voluminous tumors and generally manifest when they metastasize.

Symptoms

The essential thing is the virilism that is detected in children and women. Adult men do not recognize increased virilization.

Complementary exams

1. Excessively increased 17-ketosteroids in the urine.

Late or postpubertal forms

Symptoms

They are characterized in the female sex by the appearance in a previously normal woman of a virilization syndrome (fig. 75.8):

1. Hirsutism: development of male sexual hairs (moustache, beard, pre-tragus region, hairiness of the white line).
2. Morphological modifications: muscle development, skin thickening, acne.
3. Behavior modifications: hyperactivity, hoarse voice.
4. Virilization of sexual characteristics: atrophy of the breasts, hypertrophy of the clitoris, amenorrhea.

In the male sex, the late forms are rare and difficult to perceive, since they consist of the accentuation of the characteristics of the sex itself.

Contrasts exaggerated sexual, genital and extragenital development, with testicular atrophy.

Complementary exams

1. Increase in urinary elimination of 17-ketosteroids.
2. 17-hydroxycorticoids are normal.
3. Electrolytes and blood glucose are normal.

Etiology of adrenogenital syndrome

In young children and infants, the cause of adrenogenital syndromes is congenital adrenal hyperplasia.

It can also be produced by adrenal adenomas and carcinomas (see Fig. 75.8), but less is known about the mechanisms of production of these neoplasms. It can be assumed that there are also enzymatic defects in these tumors.

About 50% of these tumors with adrenogenital syndrome occur before 12 years of age.

ESTROGENIC HYPERCORTICISM: SYNDROME DUE TO EXCESS ESTROGEN (FEMINIZATION)

Feminization syndrome in adult men is very rare; It is due to an increase in the production of estrogens by the adrenal cortex and is almost always of tumor origin.

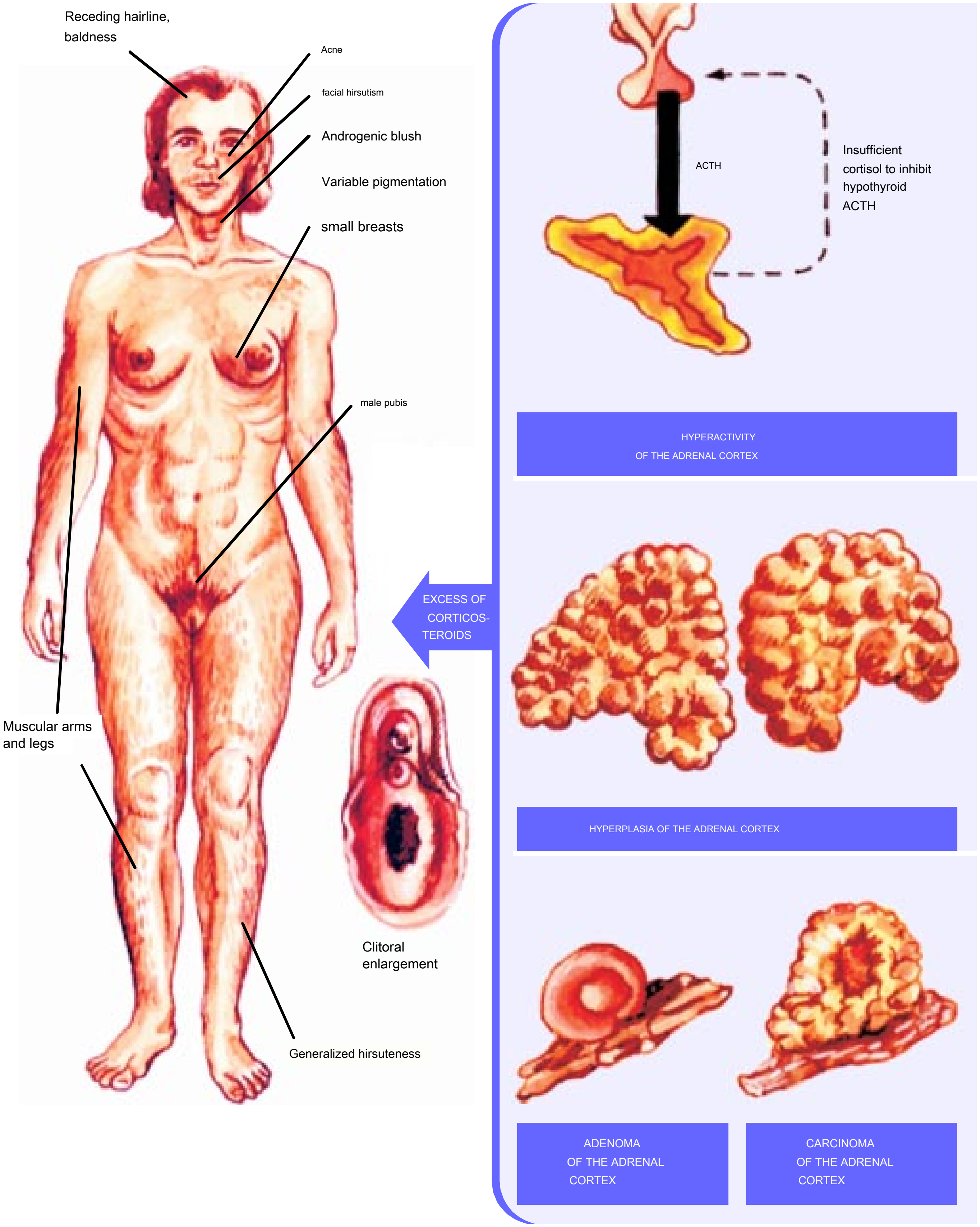

Receding hairline,
baldness
Acne
facial hirsutism
Androgenic blush
Variable pigmentation
small breasts
male pubis
Muscular arms
and legs
Clitoral
enlargement
Generalized hirsuteness
EXCESS OF
CORTICOS-
TEROIDS
ACTH
Insufficient
cortisol to inhibit
hypothyroid
ACTH
HYPERACTIVITY
OF THE ADRENAL CORTEX
HYPERPLASIA OF THE ADRENAL CORTEX
ADENOMA
OF THE ADRENAL
CORTEX
CARCINOMA
OF THE ADRENAL
CORTEX

Fig. 75.8 Adrenogenital syndrome in women.

Syndromography or positive diagnosis

Clinical picture

It is characterized by:

1. Gynecomastia, the most prominent sign.
2. Testicular atrophy.
3. Impotence.
4. Loss of sexual, genital and extragenital hair.
5. Other signs of adrenal hyperactivity, such as hyper- blood pressure.

Complementary exams

1. Hormonal:
   a) Elevation of plasma 17-beta-estradiol.
   b) Excess estrogen in the urine.
2. Imaging:
   a) Abdominal ultrasound: the adrenal tumor is observed.
   b) Computed axial tomography of the adrenal glands: shows the tumor.
   c) Pyelography and aortography also demonstrate the adrenal tumor mass.

HYPERMINERALOCORTICISM0: PRIMARY HYPERALDOSTERONISM 0 CONN

Concept

In the fairly strict sense, primary hyperaldosteronism is the constellation of clinical and chemical abnormalities that result from autonomous hypersecretion of aldosterone. The words primary and autonomous are used to separate this process from secondary hyperaldosteronism, that is, the hypersecretion of aldosterone in response to a known stimulus, such as angiotensin.

Primary hyperaldosteronism generally results from an aldosterone-secreting adenoma (Conn syndrome), but is sometimes associated with adrenal hyperplasia or morphologically normal adrenal glands (idiopathic hyperaldosteronism).

Syndromogenesis or pathophysiology

Aldosterone is responsible for 95% of the mineralocorticoid activity of the adrenal cortex. Corticosterone and deoxycorticosterone also participate in these effects.

The effects of aldosterone are (fig. 75.9):

1. Increased sodium reabsorption in the renal tubule.
2. Increased potassium excretion.

The first is accompanied by mild hypernatremia, polydipsia, increased extracellular fluid volume and blood volume, a tendency to increase cardiac output, increased general vascular resistance and arterial hypertension.

The second is accompanied by muscle weakness, paralysis due to a decrease in intracellular potassium, hypokalemia and hypokalemic alkalosis.

Excess excretion of mineralocorticoids explains the syndrome described by Conn in 1955.

Syndromography or positive diagnosis

Clinical picture (fig. 75.10)

High blood pressure is the main element of this syndrome; It is systodiastolic, generally moderate, quite stable and benign in appearance; It is manifested by headaches; The vascular impact is discrete for a long time.

The associated signs are relatively rare, but when present they suggest the diagnostic possibility:

1. Paroxysmal attacks of muscle weakness that can reach pseudoparalysis, against a background of diffuse muscular asthenia that predominates upon awakening.
2. Tetaniform crises, less rare in women (around 25% of cases) but often replaced by more common signs of neuromuscular excitability: cramps, paresthesias; especially polyuropolydipsic syndrome (diuresis of 2-5 L/24 h), much more frequent, predominantly nocturnal and resistant to pitresin.

A negative character becomes important in this clinical picture: the absence of edema.

In reality, the clinical aspects have different degrees and atypical forms are very common. Everything can be reduced to arterial hypertension, accompanied by chronic muscular asthenia and moderate polyuria. Hypertension itself can take on an unusual appearance, be accentuated by paroxysms, or even take on a malignant evolution. Hence the importance of the biological syndrome, which must be investigated in all isolated arterial hypertension, especially in a young woman.

Complementary exams

Laboratory

In the blood we find: hypokalemia, hypernatremia, alkalosis and hyperaldosteronemia.

The urine is alkaline and shows: increased K, low Na content and hyperaldosteronuria.

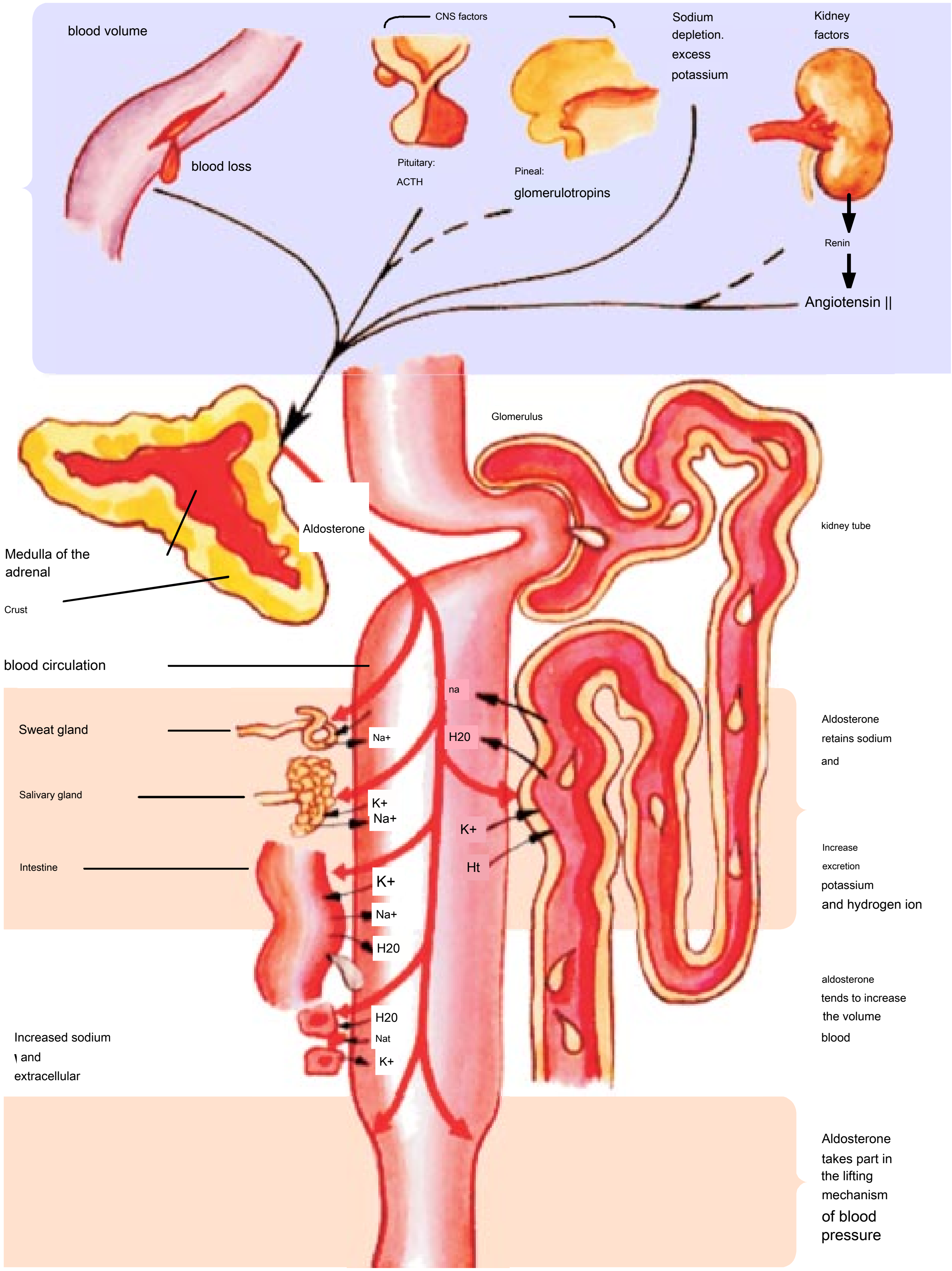

blood volume
CNS factors
Sodium
depletion.
excess
potassium
Kidney
factors
blood loss
Pituitary:
ACTH
Pineal:
glomerulotropins
Renin
Angiotensin ||
Glomerulus
Aldosterone
kidney tube
Medulla of the
adrenal
Crust
blood circulation
Sweat gland
Salivary gland
Intestine
na
Na+
H20
K+
Na+
K+
Ht
K+
Na+
H20
H20
Nat
K+
Aldosterone
retains sodium
and
Increase
excretion
potassium
and hydrogen ion
aldosterone
tends to increase
the volume
blood
Increased sodium
and
extracellular
Aldosterone
takes part in
the lifting
mechanism
of blood
pressure

Fig. 75.9 Biological action of aldosterone.

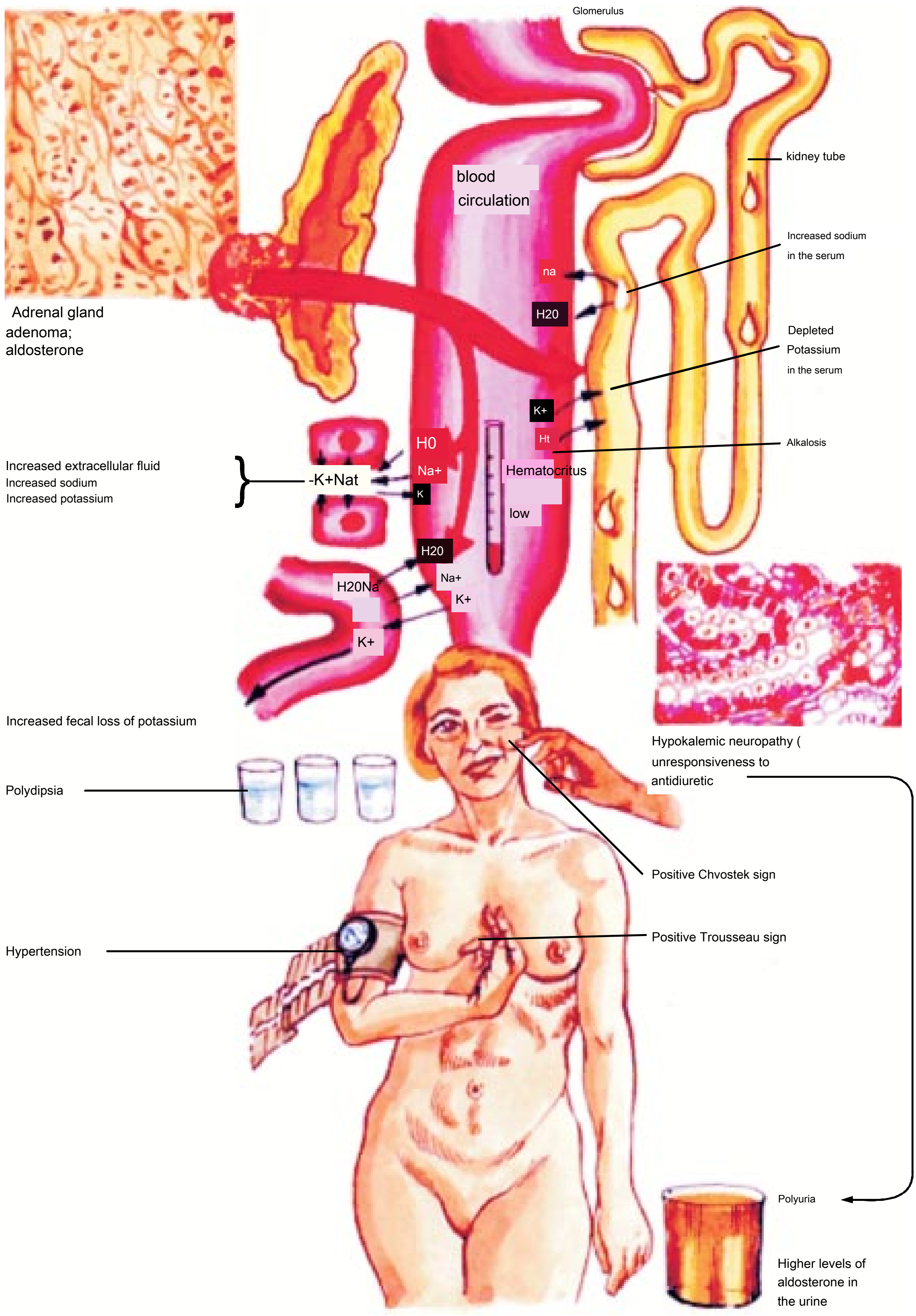

Glomerulus
blood
circulation
kidney tube
na
Increased sodium
in the serum
H20
Adrenal gland
adenoma;
aldosterone
Depleted
Potassium
in the serum
K+
Ht
Alkalosis
H0
Na+
Hematocritus
low
K
Increased extracellular fluid
Increased sodium
Increased potassium
-K+Nat
H20
Na+
H20Na
K+
K+
Increased fecal loss of potassium
Hypokalemic neuropathy (
unresponsiveness to
antidiuretic
Polydipsia
Positive Chvostek sign
Positive Trousseau sign
Hypertension
Polyuria
Higher levels of
aldosterone in
the urine

Fig. 75.10 Primary hyperaldosteronism.

Imaging

Abdominal ultrasound and CT: the tumor or adrenal hyperplasia is observed.

Etiology

A solitary adenoma is the cause of 90% of cases. Some cases are related to multiple adenomas, carcinomas or cortical hyperplasia.

In subjects under 20 years of age, bilateral, diffuse or nodular cortical hyperplasia is the most common cause.

HYPERMINERALOCORTICISM0: SECONDARY HYPERALDOSTERONISM (FIG.75.11)

It corresponds to the edematous forms that accompany: renal failure, nephrotic syndrome, heart failure, liver cirrhosis, Gestosis and, in general, all those processes where edema is a dominant symptom.

It has been shown that familial periodic paralysis is linked to excess production - by the adrenal gland - of aldosterone, or a substance similar to aldosterone, a production that would be determined by electrolyte modifications.

ACUTE ADRENAL CORTICOSINSUFFICIENCY: WATERHOUSE-FRIDERISCHSEN SYNDROME

Concept

It is a syndrome of acute or over-acute evolution, determined by a sudden cessation, absolute or relative, of adrenal cortico-adrenal functioning.

Syndromogenesis or pathophysiology

If we except cases of acute insufficiency caused by medical or surgical iatrogenesis, or those in which this occurs in chronic adrenal cortico-adrenal insufficiency, we must think, in practice, of massive bilateral adrenal hemorrhages caused by anticoagulant therapy or in the course of acute infections, especially meningococcal infections (Waterhouse-Friderischsen syndrome).

In both cases, an acute and almost total deficiency of cortisol and other hormones responsible for the circulatory system, marked hypotension, collapse and shock arise.

In Waterhouse-Friderischsen syndrome, and through the Sanarelli-Shwartzman phenomenon, a diffuse vasculopathy (consumption coagulopathy) occurs that explains the generalized petechial and hemorrhagic lesions and the consequent glandular destruction, with the coexistence of a meningeal syndrome.

Syndromography or positive diagnosis

Clinical picture (fig. 75.12)

It differs in part depending on the cause of the clinical picture, but for practical purposes, in cases of sepsis the common infectious symptoms must be added. The symptoms are abrupt in onset when rapid, insidious glandular destruction occurs in less severe cases or with discrete adrenal reserves.

General symptoms

Asthenia and adynamia, which contrast with restlessness and irritability; diffuse petechiae and ecchymosis, in Waterhouse-Friderischsen syndrome.

Cardiocirculatory symptoms

Tachycardia, hypotension and death from shock.

Nervous symptoms

Headaches, restlessness, delirium, seizures, confusion, obtundation and coma.

Gastrointestinal symptoms

Abdominal pain of various locations, which can be confused with acute abdominal surgical symptoms; nausea, vomiting, diarrhea. Sometimes the patient reports sudden, abrupt pain, accompanied by muscle contracture in the costovertebral angle (Rogoff's sign).

Complementary exams

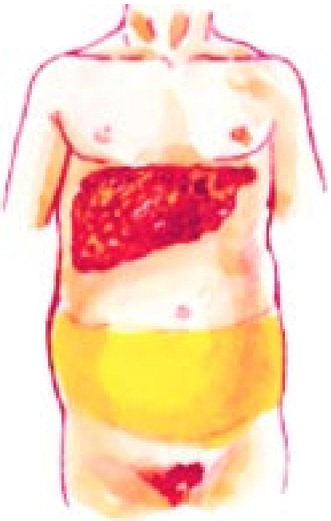
1. Hemoconcentration with increased hematocrit: determines a normal natremia, when in reality there is hyponatremia with hyperkalemia; The Na/K ratio decreases and, from a normal index of 30, reaches approximately 20.
2. Increased elimination of sodium through urine.
3. Determination of the plasma cortisol rate. It is important, but sometimes impractical, due to the speed and severity of the clinical picture,
4. Hypoglycemia and eosinophilia.

In Waterhouse-Friderischsen syndrome the blood data that accompany consumption coagulopathies are found.

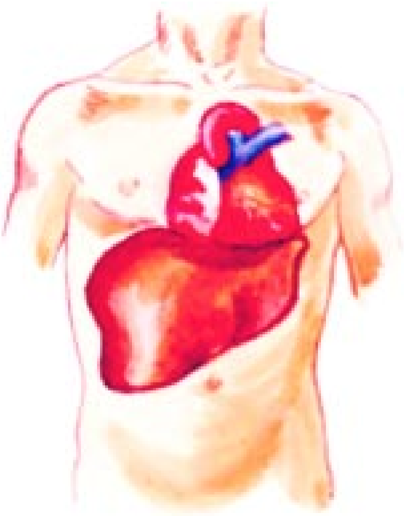
In practice, given the clinical picture, the verification of hypoglycemia, hyponatremia and hyperkalemia is largely sufficient to impose extremely urgent therapy.

Etiology

1. Acute crisis that develops into chronic, generally latent, adrenal insufficiency.



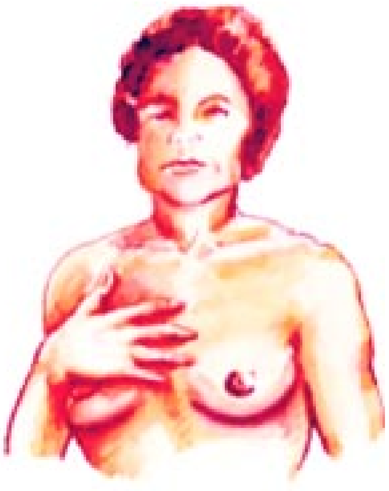
Cirrhosis of the liver with ascites



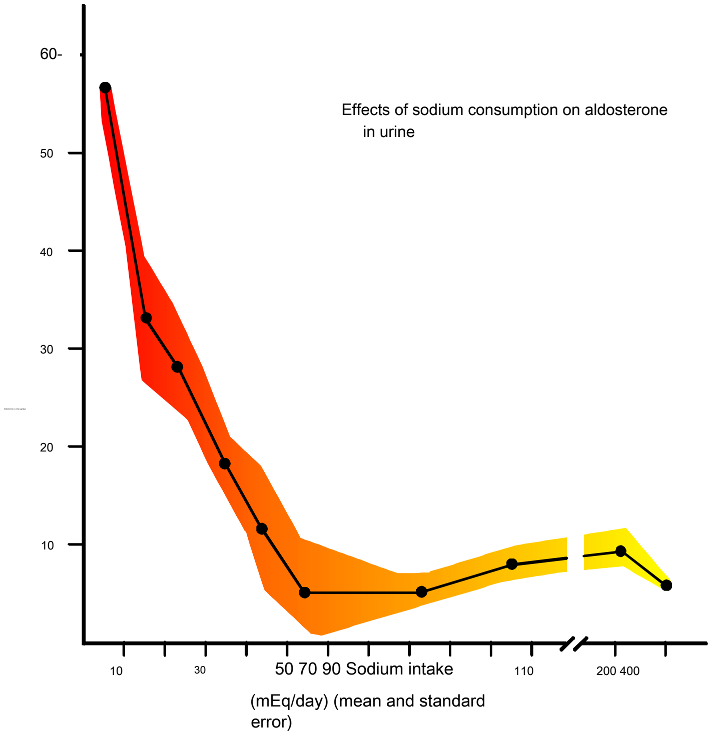
Heart disease (failure)

Malignant hypertension

Nephrosis



Cyclic idiopathic edema



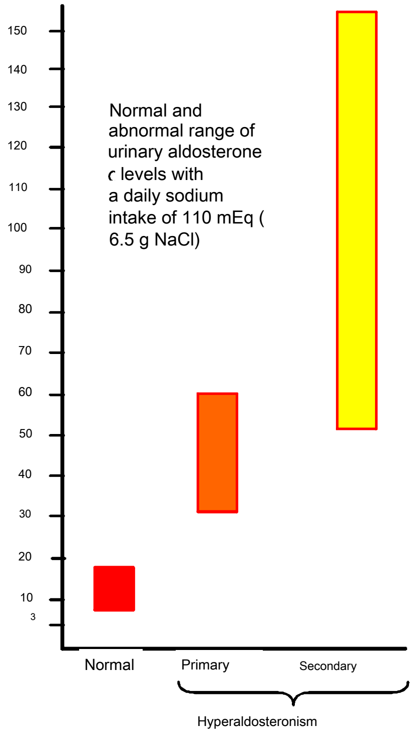


Fig. 75.11 Secondary hyperaldosteronism

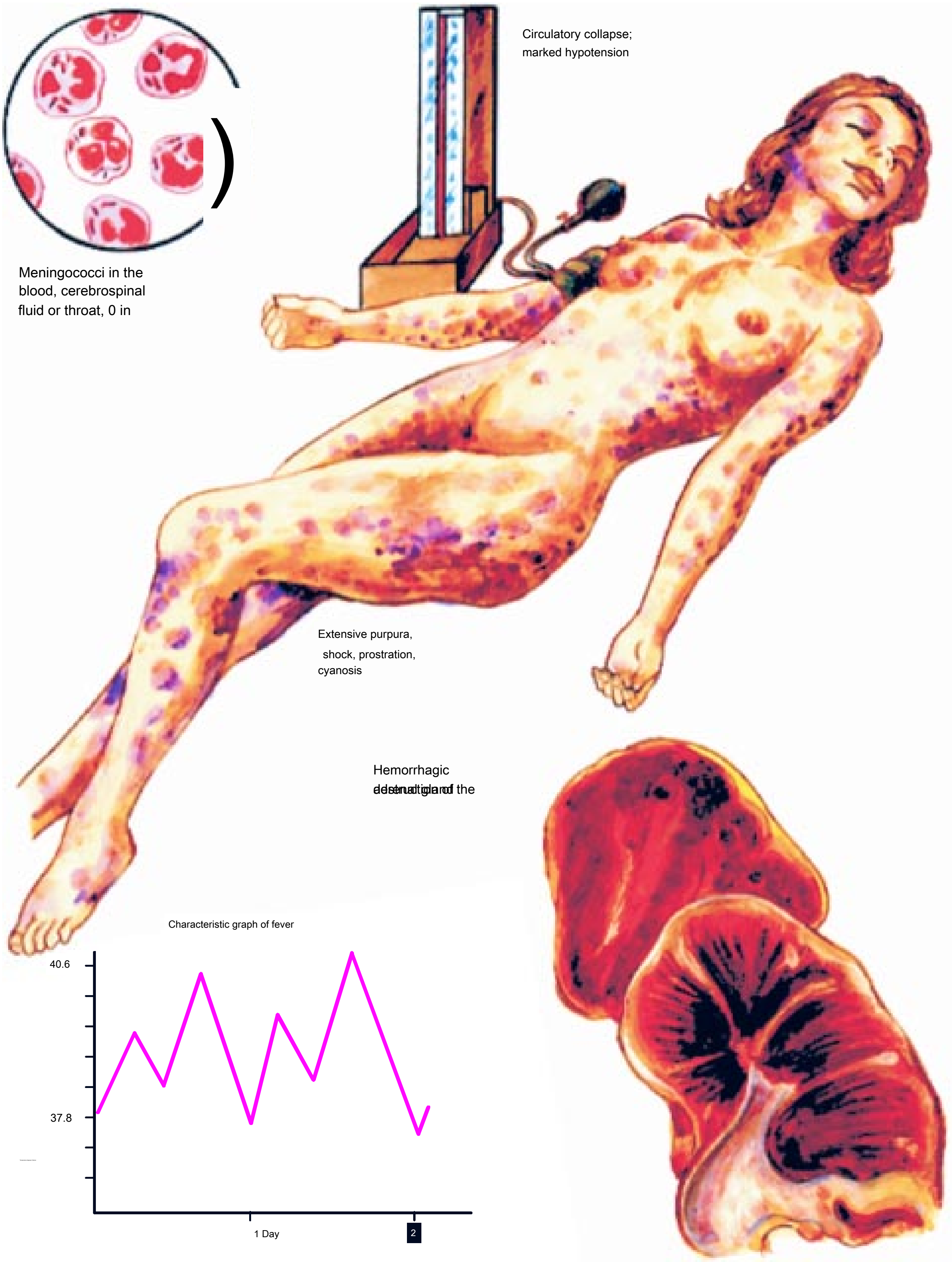

Circulatory collapse;
marked hypotension
Meningococci in the
blood, cerebrospinal
fluid or throat, 0 in
Extensive purpura,
shock, prostration,
cyanosis
Hemorrhagic
destruction of the
adrenal gland
Characteristic graph of fever
40.6
37.8
1 Day
2

Fig. 75.12 Acute adrenal corticosteroid insufficiency.

2. Infectious processes: severe diphtheria, streptococcus, pneumococcus. The so-called Waterhouse-Friderischsen syndrome of meningococcal etiology is observed with bacteremia and positive blood culture. It is known as adrenal apoplexy.
3. Hemorrhages: due to anticoagulant treatments, inflammation and necrosis.
4. Thrombosis.
5. Surgical resection.
6. Abrupt withdrawal of glucocorticoids after their intense and prolonged administration, and exposure to stress.

CHRONIC ADRENAL CORTICOSINSUFFICIENCY:

ADDISON'S DISEASE

Concept

It is a chronic syndrome, characterized by a progressive decrease in adrenal activity due to a bilateral and gradual destruction of the adrenal glands.

Syndromogenesis or pathophysiology (Fig. 75.13)

As a result of the corticoadrenal deficiency, the production of all the hormones that originate in said cortex is decreased. Therefore, there is a deficiency of the mineralocorticoid hormones that regulate the balance of electrolytes Na and K, externalizing their deficiency through a decrease in the tubular reabsorption of sodium, responsible for subsequent hyponatremia (less than 142 mEq/L) with dehydration and increased reabsorption of K with hyperkalemia.

Hypoglycemic manifestations are attributed to the secretory decrease in cortisol and its derivatives (glucocorticoids), due to poor glucose absorption at the intestinal level and deficit in gluconeogenesis (deposition of glycogen from proteins).

Sexual symptoms are linked to a decrease in androgenic hormones (nitrogen hormones).

In those conditions that also destroy the adrenal medulla, such as tuberculosis, symptoms linked to the lack of adrenaline and noradrenaline are added.

In general, the clinical manifestations should be directly attributed to the loss of hormones that regulate hydrosaline and sugar metabolisms. As a result of the loss of some of these corticoadrenal hormones, there will be, due to lack of feedback control, a compensatory increase in pituitary trophic hormones, particularly ACTH. Regarding melanoderma (darkening of the skin), so important in the picture clinical presentation of Addison's disease, it is assumed that it is also produced by the lack of pituitary inhibition, which would lead to hyperproduction of the melanophore hormone, responsible for pigmentation. Currently, the pigmentogenic role of ACTH (which, on the other hand, has a certain number of common amino acids) has been demonstrated with melanocytostimulating peptides and it has been possible to specify that pigmentation appears in Addisonian from a plasma level of ACTH between 1.5 and 2 mU/dL.

Syndromography or positive diagnosis

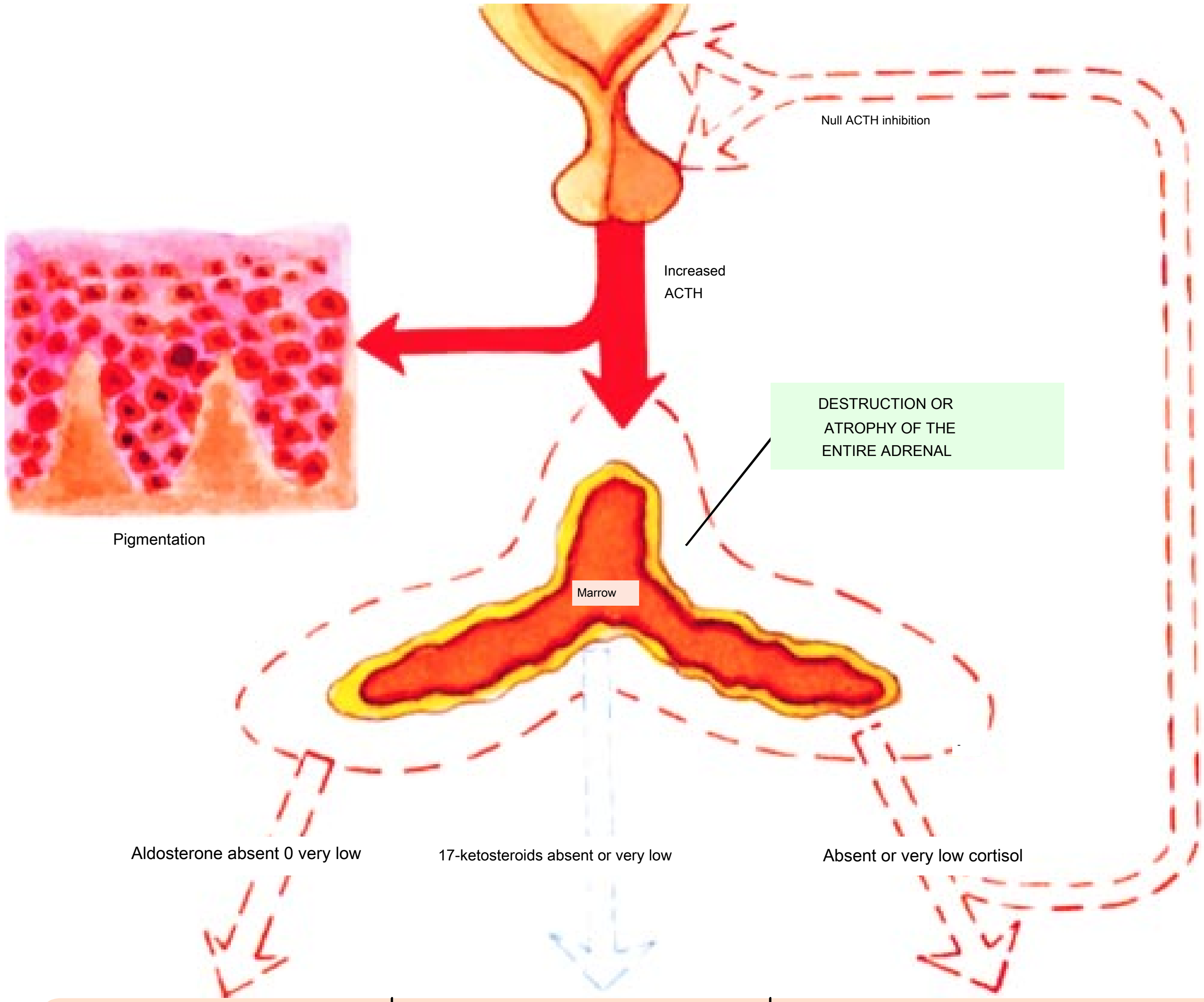
Clinical picture (fig. 75.14)

It includes five main signs:

1. Exertional asthenia. Increased fatigue due to fatigue due to muscular exercise, which can lead to true exhaustion, without any neurological sign.
2. Melanoderma. Very specific when it dyes the skin a sepia brown tone, more maintained in certain areas: exposed or normally pigmented regions, irritated areas, scars, palmar flexion and extension folds of the fingers, dirty-looking nipples. It also sometimes reaches the mucous membranes (buccal, genitals) in the form of slate spots and can even affect the pharynx. The absence of this symptom calls into question the diagnosis of Addison's disease.
3. Gastrointestinal disorders. They are often reduced to anorexia, but the unexpected appearance of nausea or vomiting, diarrhea or abdominal pain very often announces decompensation.
4. Weight loss. It is almost constant and weight fluctuations are a faithful manifestation of evolution.
5. Arterial hypotension. It is less constant, and often very moderate outside of crises, although it can be exaggerated in orthostatism. Without being essential for the diagnosis, it has a high prognostic value and its accentuation is the best alarm signal.

In addition, there may be other symptoms:

1. Manifestations of hypoglycemia.
2. Genital disorders, especially impotence in men, while in women amenorrhea and sterility are rare.
3. Psychological disorders, with phases of depression that alternate with anxious instability.
4. Arthralgias or paroxysmal myalgias, aggravated by mineralocorticoids and canceled by cortisone.


Null ACTH inhibition
Increased ACTH
Pigmentation
DESTRUCTION OR ATROPHY OF THE ENTIRE ADRENAL
Marrow
Aldosterone absent 0 very low
17-ketosteroids absent or very low
Absent or very low cortisol

Clinical features	Hypotension	Reduced pubes Loss of muscle substance Loss of libido	Pigmentation Weakness slow cerebration Achlorhydria Anorexia Nausea
Blood	Nat blood volume — Low HC0, IC } — Bass K+ — High Na/ — Less than 30 K Cretinin — Elevated		Anemia Normocytic, normochromic: Hemoglobin — -Low Neutropenia Relative lymphocytosis Moderate hypoglycemia 17-hydroxycorticoids — -Low Inadequate increase after ACTH
Urine	Aldosterone — Low (Less than 5 mg/24 h)	12- ketosteroids — Bass (Less than 5 mg/24 h)	17-hydroxycorticoids — -Basses Less than 3 mg/24 h Inadequate increase after

Fig.75.13 Pathophysiology of Addison's disease.

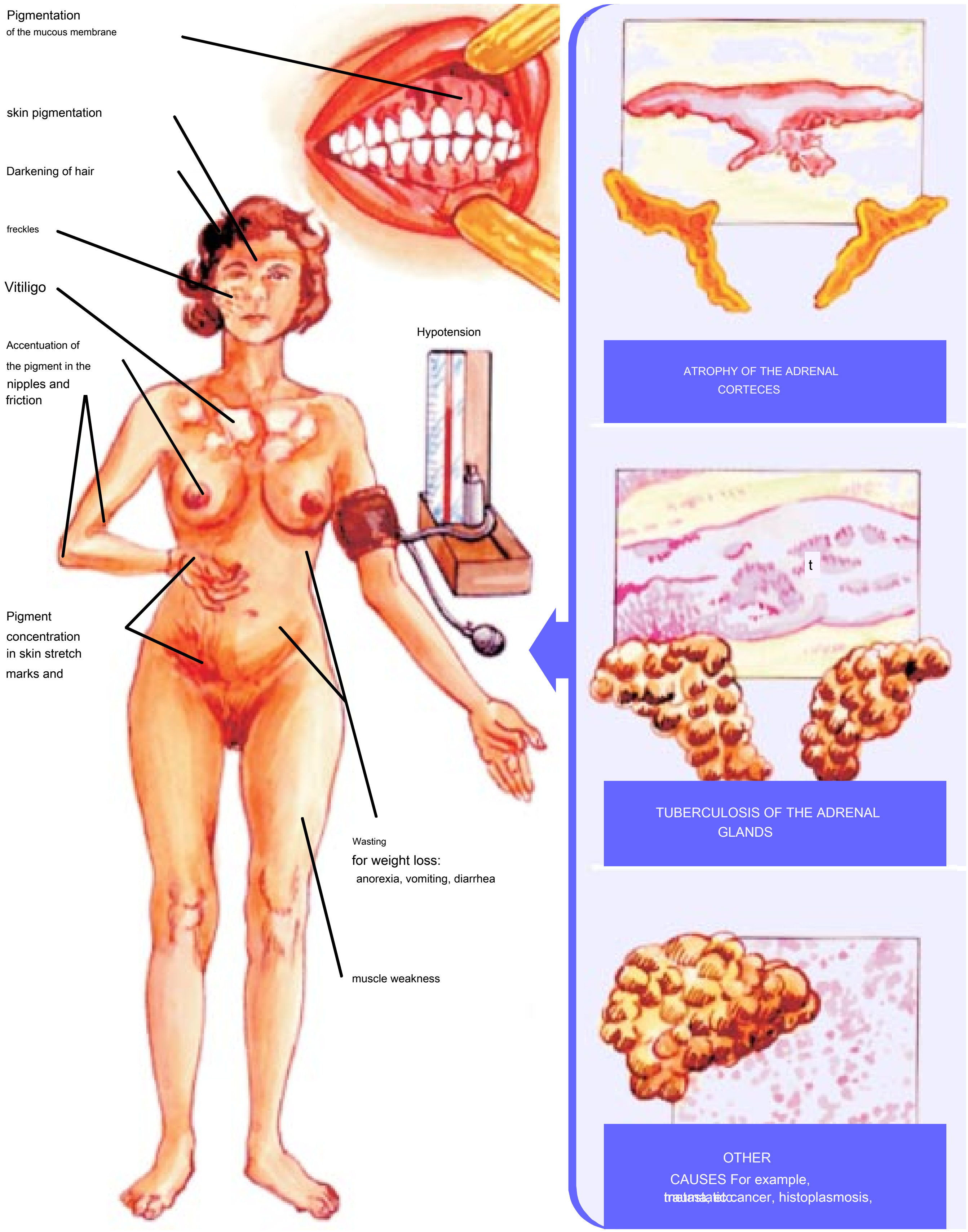

Pigmentation
of the mucous membrane
skin pigmentation
Darkening of hair
freckles
Vitiligo
Accentuation of
the pigment in the
nipples and
friction
Pigment
concentration
in skin stretch
marks and
Hypotension
Wasting
for weight loss:
anorexia, vomiting, diarrhea
muscle weakness
ATROPHY OF THE ADRENAL
CORTECES
t
TUBERCULOSIS OF THE ADRENAL
GLANDS
OTHER
CAUSES For example,
trauma, metastatic cancer, histoplasmosis,

Fig. 75.14 Chronic adrenocortical insufficiency.

Complementary exams

1. Tests that show a decrease in mineralocorticoids:

   a) Increased elimination of chlorine, sodium and water through urine.

   b) Hyponatremia, hypochloremia and hyperkalemia.

   c) 17-oxysteroids are low (normal: 4-6 mg/24 h in men; 1-3 mg/24 h in women).

2. Tests that show a decrease in glucocorticoids:

   a) Hypoglycemia.

   b) Anemia, leukopenia, eosinophilia and lymphocytosis.

   Decrease in basal plasma cortisol to 10%.

   d) Elevation of plasma ACTH.

   e) ACTH stimulation test (the most important).

   Plasma cortisol is not elevated.

3. Exams that show the decrease in androgens:

   a) Elevation of azotemia.

   b) Decrease in the excretion of 17-ketosteroids in the urine.

Etiology (see Fig. 75.14)

Unlike what occurs in acute insufficiency, the complete picture of Addison's disease only manifests itself when the noxa has produced the destruction or involution of 90% of the two corticoadrenals.

Primary or idiopathic atrophy (60%), tuberculosis and amyloidosis constitute 99% of the causes found.

Metastatic neoplasms, mycoses, histoplasmosis and other causes, such as fatty degeneration, venous thrombosis and arterial embolism, are very rare.

Autoimmune mechanisms have been involved in the so-called idiopathic atrophy, due to the increasingly frequent finding (especially in the United States) of anti-adrenal antibodies in the serum and the coexistence with other conditions such as Hashimoto's thyroiditis.

Adrenal insufficiency can sometimes be secondary to hypopituitarism.

MEDULAR HYPERFUNCTION SYNDROME (PHEOCHROMOCYTOMA)

Concept

It is a clinical-biological syndrome determined by hyperfunctioning of the adrenal medulla.

Its usual etiopathogenic basis is a hyperfunctioning neoplasia, almost always benign, that comes from pheochromocytes: pheochromocytoma.

The individualization of this syndrome is of the greatest importance, since the removal of the neoplasia allows the cure of hypertension, which is the fundamental manifestation of the clinical picture.

Syndromogenesis or pathophysiology (Fig. 75.15)

The common manifestations of the syndrome are related to the production of increased amounts of catecholamines. Very exceptionally, in cases of pheochromocytoma, the physical presence of the tumor or tumors can justify the clinical picture, since the same

mos are small. These tumors produce excessive amounts of norepinephrine and adrenaline in a paroxysmal or constant manner. When they release large amounts of norepinephrine in excessive and paroxysmal sympathetic stimulation, their symptoms are reminiscent of paroxysmal arterial hypertension, against a background of normal or slightly increased blood pressure. This paroxysmal course results in the production of hypertensive crises that can last minutes or hours. When, on the contrary, the release of norepinephrine is discrete and sustained, a hypertensive condition is generated that is similar to essential arterial hypertension, with permanently increased values of the systolic and diastolic figures.

Pheochromocytoma is a rare tumor. It is estimated that 0.1% (one per thousand) of high blood pressure are caused by it.

Syndromography or positive diagnosis

Clinical picture (fig. 75.16)

Pheochromocytoma essentially causes tension disorders, which can take three forms:

paroxysmal arterial hypertension, permanent arterial hypertension and orthostatic

Paroxysmal arterial hypertension crises are the most characteristic, but they cover only a quarter of the cases. They begin suddenly with intense discomfort with chills, then headaches appear in the ascending wing, which end in an intense headache, while the paleness is extreme and the sweating is profuse. However, tachycardia is observed (followed by bradycardia of vagal origin) and, above all, a sudden rise in blood pressure to levels that immediately guide the diagnosis. Between these paroxysms (from a few minutes to several hours), with a sudden ending, the examination is perfectly normal; Tolerance can remain good for a long time, but in some cases the attacks become more frequent and severe and, sometimes, lead to permanent high blood pressure. They can also lead to vascular complications or adrenaline shock with lumbar pain and imperceptible tension, which may or may not be accompanied by sudden death.

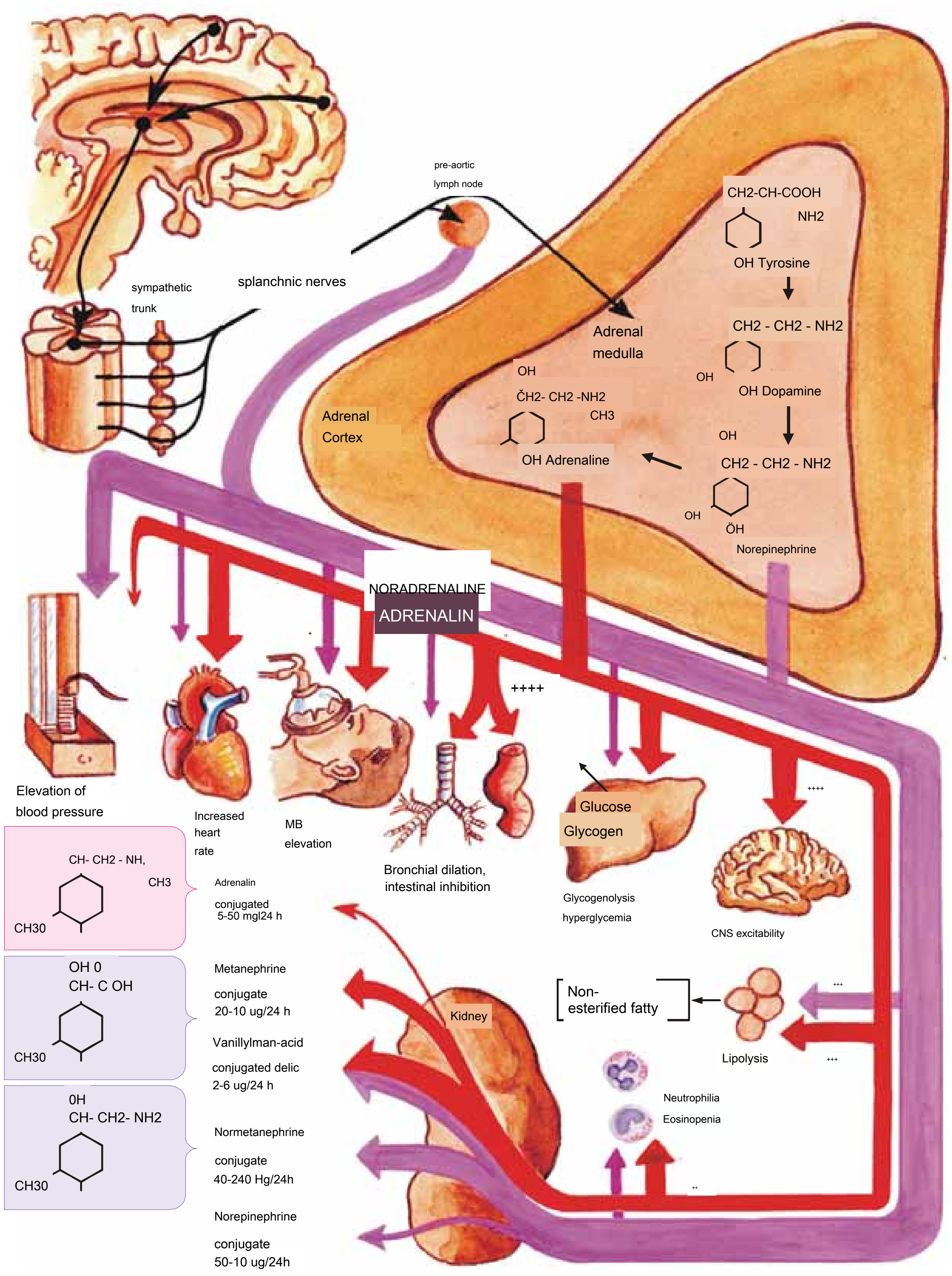

pre-aortic
lymph node
CH2-CH-COOH
NH2
OH Tyrosine
sympathetic
trunk
splanchnic nerves
Adrenal
medulla
CH2 - CH2 - NH2
OH
OH Dopamine
OH
ČH2- CH2 -NH2
CH3
Adrenal
Cortex
OH Adrenaline
OH
CH2 - CH2 - NH2
OH
ÖH
Norepinephrine
NORADRENALINE
ADRENALIN
++++
Elevation of
blood pressure
Increased
heart
rate
MB
elevation
Bronchial dilation,
intestinal inhibition
Glucose
Glycogen
Glycogenolysis
hyperglycemia
++++
CNS excitability
CH- CH2 - NH,
CH3
CH30
Adrenalin
conjugated
5-50 mgl24 h
OH 0
CH- C OH
CH30
Metanephrine
conjugate
20-10 ug/24 h
Vanillylman-acid
conjugated delic
2-6 ug/24 h
Kidney
Non-
esterified fatty
+++
Lipolysis
+++
Neutrophilia
Eosinopenia
++
0H
CH- CH2- NH2
CH30
Normetanephrine
conjugate
40-240 Hg/24h
Norepinephrine
conjugate
50-10 ug/24h

Fig. 75.15 Pathophysiology of pheochromocytoma.

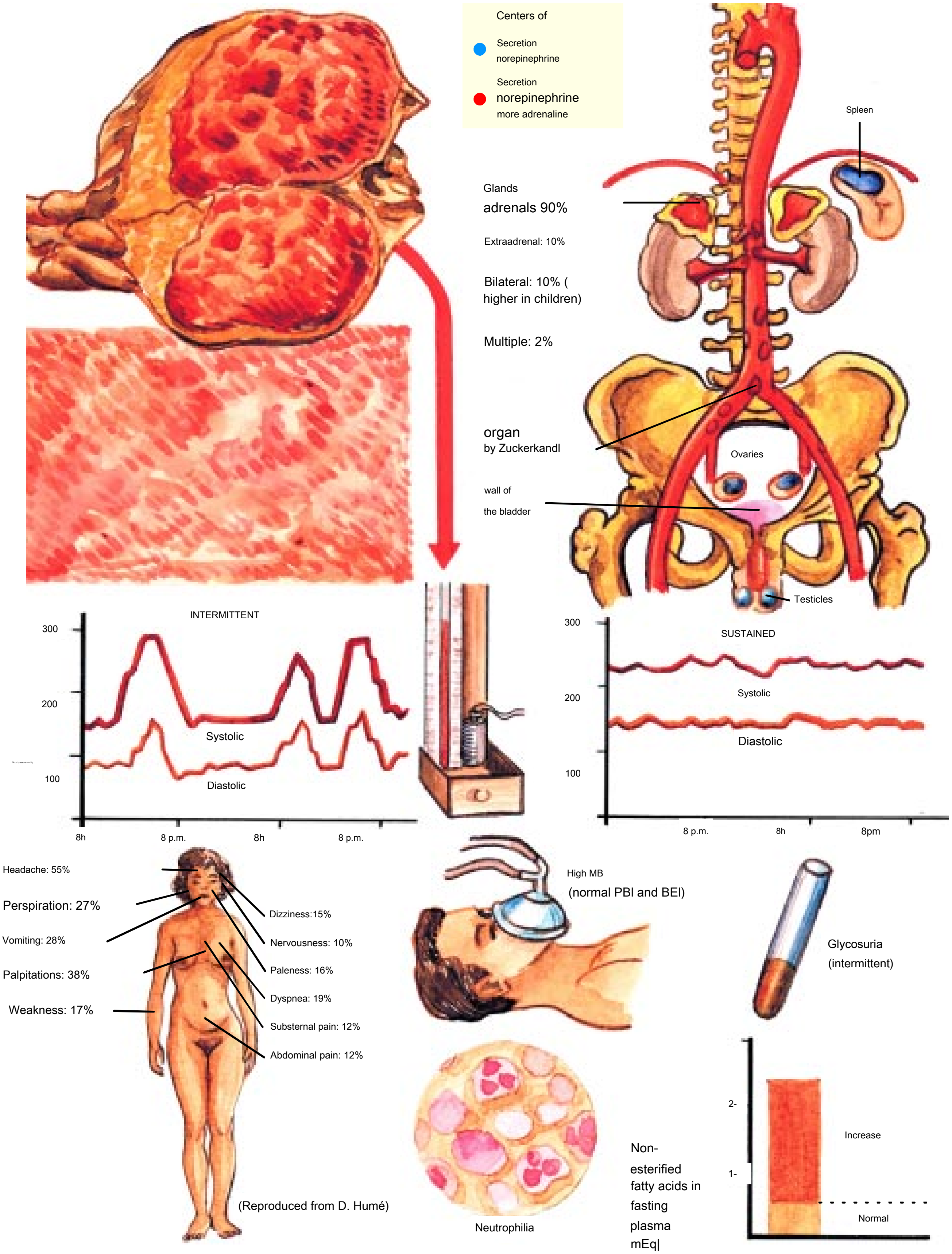

Centers of
● Secretion norepinephrine
● Secretion norepinephrine more adrenaline
Spleen
Glands
adrenals 90%
Extraadrenal: 10%
Bilateral: 10% (higher in children)
Multiple: 2%
organ by Zuckerkandl
Ovaries
wall of the bladder
Testicles
INTERMITTENT
300
200
100
Systolic
Diastolic
8h
8 p.m.
8h
8 p.m.
SUSTAINED
300
200
100
Systolic
Diastolic
8 p.m.
8h
8pm
Headache: 55%
Perspiration: 27%
Vomiting: 28%
Palpitations: 38%
Weakness: 17%
Dizziness:15%
Nervousness: 10%
Paleness: 16%
Dyspnea: 19%
Substernal pain: 12%
Abdominal pain: 12%
(Reproduced from D. Humé)
High MB
(normal PBI and BEI)
Glycosuria (intermittent)
Neutrophilia
Non-esterified fatty acids in fasting plasma mEq|
2-
1-
Increase
Normal

Fig. 75.16 Clinical picture and etiology of adrenal medullary hyperfunction (pheochromocytoma).

Permanent arterial hypertension is, in fact, more frequent (60-70% of cases), but some nuances may draw attention: instability of this hypertension and existence of proven paroxysms, on the occasion of sudden headache or sudden tachycardia. Youth, the drop in blood pressure in orthostatism, the severity of retinal lesions and the variability of kidney involvement are other arguments that suggest signs of the pheochromocytoma.

However, sometimes this permanent hypertension is very banal.

Finally, orthostatic hypotension can, although very rarely, be revealing and even cause true collapses.

Other symptoms associated with one or another of these tension modifications help in the diagnosis: sweating crises, even in the absence of hypertensive access, a constant sign according to Mornex; slimming; feverish outbreaks; psychological disorders with anxiety; abdominal painful crises of the solar type, etc.

Complementary exams

1. Catecholamines in urine: increase above normal levels.
2. Determination of vanillylmandelic acid (VMA) in urine: elevated. Normal figures: 1-8 mg/24 h. The finding of a figure greater than 10 mg/24 h is very suspicious.
3. Epinephrine and norepinephrine in blood and urine: elevated.
4. Functional tests:
   a) In the hypertension phase: regitin test (drop in blood pressure, more than 35 mm Hg for the maximum and 25 mm Hg for the minimum).
   b) In the normotension phase: histamine or tyramine test (triggering hypertensive crisis).
5. Abdominal ultrasound. To detect adrenal tumor, which is generally unilateral.
6. CT scan of the adrenal glands. It is the imaging test of choice.
7. Renal arteriography. It should be used when there are doubts with other diagnostic procedures.

Etiology (er fig. 75.16)

Since Frankel reported the first tumor in 1886, thousands of cases have been reported in adults and children to date. This tumor affects the right adrenal in 48% of cases, the left in 33% and is bilateral in 9%, which represents 90% of cases that occur in the adrenal glands.

Pheochromocytoma can be found in other chromaffin tissues, along the abdominal pre-aortic sympathetic chain and especially in the so-called Zuckerkandl organ, located at the bifurcation of the aorta.

It has also been reported in the spleen, ovaries, testes, bladder and mediastinum.

TUMORAL SYNDROME

The small size and deep location of the adrenal glands explain that adrenal tumors rarely cause symptoms and signs that can be detected during physical examination.

Syndromography or positive diagnosis

Clinical picture

When they reach sufficient size, these neoplasms can attract the attention of the patient and the doctor due to the deformity they produce in the flank or hypochondrium where they are located. What bothers the patient the most are the lumbar painful manifestations, radiating laterally or towards the corresponding lower limb.

During the physical examination, signs typical of tumors of the lumbar fossa are obtained.

Large neoplasms are generally malignant and are accompanied by the usual manifestations of cachexia and metastasis, mainly in bones, bone marrow or different viscera.

Complementary exams

1. Abdominal CT scan. It allows the enterochromaffin tumor to be revealed.
2. Exploratory laparotomy. It is performed when there is a fairly well-founded suspicion of a tumor and the CT scan offers doubts.

76

ENDOCRINOMETABOLIC SYSTEM. HYPERGLYCEMIC AND HYPOGLYCEMIC SYNDROMES

General considerations

The rate or amount of glucose in the blood is called glycemia. Normally it is 60-110 mg% or its equivalent, 3.3-6.2 mmo/L.

Let us remember that 1 mmol of glucose a/Le is equivalent to 18 mg%. So if the figure is to be expressed in mmol/L, just divide the amount in mg% by 18. When it rises above 120 mg% (6.7 mmol/L) it is called hyperglycemia, and when it falls below 40 mg% or its equivalent 2.2 mmol/L, it is called hypoglycemia.

Factors that regulate glycemia. Hemoglucoregulatory systems maintain the blood glucose level constant between 60 and 80 mg% (3.3-4.5 mmol/L). After ingestion of food, there is a temporary increase in blood glucose, due to intestinal absorption, to return to normal by virtue of the physiological mechanisms that regulate glucose.

These factors are:

1. Intestinal absorption. The impairment of intestinal absorption produces a decrease in blood glucose. We frequently see this in intestinal malabsorption syndromes and chronic diarrhea.
2. Hepatic glycogenesis and gluconeogenesis. If the formation of liver glycogen (glycogenesis) is hindered, glucose will be found circulating in the blood without being deposited in the liver. If, on the other hand, there is a marked increase in glycogenesis, blood glucose levels tend to decrease, since glycogen is formed by the liver at the expense of blood glucose.
3. The stimuli that increase glycogenesis are: hyperglycemia, insulin and hormones of the adrenal cortex.
4. Glycogenesis is decreased in liver diseases and in cases of hypoinsulinism.
5. Hepatic gluconeogenesis occurs when blood sugar levels drop and the liver cannot make glycogen with blood glucose, but it can make glycogen at the expense of amino acids or fatty acids.
6. Hepatic glycogenolysis. It occurs under normal circumstances to maintain normoglycemia in accordance with tissue requirements; It is intensified, therefore, by increased glucose consumption, by the administration of sympathomimetic substances such as adrenaline, by hypoinsulinism in diabetes mellitus.

of man or experimental of animals, and finally, in cases of hyperadrenalism, adrenal hypercorticism, hyperthyroidism and anterior hyperpituitarism, whose hormones all tend to mobilize hepatic glycogen giving rise to an increase in blood glucose.

7. Tissue use. The consumption that occurs in the tissues also regulates blood glucose.
8. Renal excretion. When the blood glucose level rises above the renal threshold, glucose is eliminated (glycosuria).
9. Muscular metabolism Muscular activity tends to reduce blood glucose.
10. Pancreas. In addition to participating through digestive ferments in the digestion of starches, the endocrine pancreas, through insulin and glucagon, constitutes an element of vital importance in glucoregulation.

In saccharin diabetes, a genetic metabolic disorder characterized by a lack (absolute or relative) of insulin, a disorder occurs in the use of carbohydrates and alterations in lipid and protein metabolism that lead to hyperglycemia, glycosuria, hypercholesterolemia and frequent ketoacidosis. Metabolic disorders are usually controlled with the use of insulin or oral hypoglycemic agents.

This hereditary disease must be differentiated from diabetes acquired by pancreatic lesions or by surgical removal, where an important part of the pancreas and its islets are destroyed or eliminated, and is also different from the so-called endocrine diabetes of hyperthyroidism, hyperadrenalism, acromegaly, pheochromocytoma, etc.

Insulin produced in the B cells of the islets of Langerhans acts as a factor that promotes glucose phosphorylation, opposing the contrainsular system (glucagon, pituitary, adrenaline and adrenaline). As a result of its physiological action, insulin:

a) increases the ease of glucose penetration into cells and its tissue use,

b) increases glycogenesis,
c) inhibits gluconeogenesis,
d) decreases blood glucose,
e) facilitates hepatic lipogenesis,
f) exerts a favorable effect on the deposition of albumin in the muscle (Mirsky effect),

g) due to its anabolic action, it facilitates growth.

In the clinic, the most frequent pathophysiological disorders are: the decrease in insulin secretion, causing a hyperglycemic syndrome, or the clinical picture determined by an excess of insulin secretion (due to hyperplasia, benign or malignant insulinoma), which causes the hypoglycemic syndrome.

Glucagon produced in islet cells is a hyperglycemic and glycogenolytic hormone. More than an antagonistic hormone, it maintains an interaction with insulin in determining normoglycemia.

11. Pituitary gland (partly through ACTH), adrenal glands and thyroid gland.
12. Both the central nervous system and the parasympathetic vegetative system (through the insular system) or sympathetic system (through the release of adrenaline, a chemical mediator in the sympathetic nerve endings) clearly influence blood glucose.
13. Pregnancy and false pregnancy in women receiving exogenous estrogens. Many latent diabetic patients can become manifest through the administration of oral contraceptives (contraceptive medications).

HYPERGLYCEMIC SYNDROME

CONCEPT

It is the humoral state in which blood glucose levels are above normal.

A high basal glycemia (fasting), a postprandial glycemia (2 hours after a main meal) also elevated, and an altered glucose tolerance test (GTT), diabetic type, allow establishing the diagnosis of disturbance of carbohydrate metabolism.

However, we must point out that hyperglycemia, with or without glycosuria, is not synonymous with diabetes. Analogous states may exist due to nervous, psychogenic, emotional, hepatic, endocrine or iatrogenic causes.

SYNDROMOGENESIS OR PHYSIOPATHOLOGY AND POSITIVE DIAGNOSIS

We will fundamentally analyze the pathophysiology and clinical picture of diabetes mellitus as it is the most frequent cause of hyperglycemia and persistent glycosuria.

try

The hyperglycemia of diabetes mellitus is due to absolute or relative insulin insufficiency. When missing

This hormone decreases the use of glucose by peripheral tissues, muscles and adipose tissue; The liver, in response to peripheral demand, increases glycogenolysis, which further increases the blood glucose level. When this figure exceeds the renal threshold, which is approximately 180 mg/dL (10 mmolL), excretion begins through this route, which carries large amounts of fluid due to its osmotic power, causing polyuria with glycosuria. This results in the need to drink water, which causes marked thirst and gives rise to polydipsia.

On the other hand, since the tissues cannot adequately use glucose, they first destroy the lipids of the fat reserves and subsequently the proteins, since although there is a large amount of glucose in the blood, the tissues continue to be hungry for glucose and this has several consequences:

1. Feeling of hunger, that is, polyphagia, to try to ingest energy substances.
2. Ketoacidosis due to accelerated lipid metabolism, which can lead to diabetic coma.
3. Increased gluconeogenesis in the liver from the products of lipoprotein metabolism.
4. In most cases, an increase in blood lipids, including cholesterol.

All these alterations cause:

- Muscular and mental asthenia, with a feeling of tiredness, weakness and lack of concentration.
- Weight loss due to weight loss, which is established as the condition progresses and which can become important, since progressive dehydration is added.

These are the main or most frequent symptoms in diabetes mellitus; But if the disease reaches great intensity before being diagnosed or is not adequately controlled after becoming aware of it, it gives rise to a good number of complications, with its characteristic symptomatic procession.

The most important ones are:

1. Vascular manifestations. Generalized atherosclerosis is common, in relation to the increase in blood lipids or other mechanisms and whose clinical manifestation depends on the affected vessel: coronary artery disease and myocardial infarction are common; stroke apoplecticus (cerebral); and when they affect the arteries of the lower limbs, intermittent claudication, arteritis and the feared diabetic gangrene occur.

   When the arterioles are affected, the so-called diabetic angiopathy (microangiopathy) occurs, which usually lead to kidney lesions that cause arterial hypertension and determine kidney failure syndromes, including Kimmelstiel-Wilson syndrome or intercapillary glomerulosclerosis and visual disorders, especially of the retina, as an expression of the so-called diabetic retinitis. Sometimes, diabetics go to the ophthalmologist before the general practitioner. These disorders can take the form of microaneurysms or proliferative retinitis.

2. Neurological manifestations. Neuritis and neuralgia; the so-called diabetic neuropathy and the plantar perforating disease that is extremely rebellious and tenacious to therapy.
3. Skin manifestations. Annoying itching is often found on the skin and in women, vulvar itching.
   Skin infections are common: folliculitis, dermatitis, boils and carbuncles, which for some constitute cutaneous diabetes.
   Due to the existing dyslipidemia, it is common to find so-called xanthomnas on the skin.
4. Infections in general, including pyelonephritis, are very common in diabetics.

COMPLEMENTARY EXAMS

They serve to confirm diabetes mellitus:

1. Glycosuria in fasting or postprandial urine (1 hour after one of the main meals) by the classic Benedict test or by more sensitive and specific enzymatic methods.
2. Fasting blood test (hyperglycemia).
3. Blood glucose 2 hours after a meal, postprandial hyperglycemia.
4. Oral glucose tolerance test (PGT).
5. Intravenous glucose tolerance test.

For the interpretation of these tests, the Clinical Laboratory texts can be consulted.

Let us also remember that hyperglycemia can have other origins and in these cases more specialized tests will be used.

ETIOLOGY

We will summarize the causes that determine hyperglycemia, some of which have already been pointed

1. Dietary hyperglycemia. It occurs in subjects who eat large amounts of carbohydrate-based foods. The interrogation helps to establish the diagnosis, and the restriction to an adequate diet normalizes it.
2. Hyperglycemia due to pituitary-adrenal predominance. It occurs in syndromes of hyperfunction of the anterior lobe of the pituitary or adrenal glands (see acromegaly, Cushing's, hyperal-

dosteronism, pheochromocytoma, etc.). It is called steroid or contrainsular diabetes, and is characterized by: insensitivity to insulin, benignity due to the absence of ketosis, easy reduction of hyperglycemia by fasting or appropriate diet, negative nitrogen balance.

This syndrome can also be found in patients treated with excess ACTH and in patients undergoing treatment with corticosteroids.

3. Hyperglycemia due to hyperadrenalism. It occurs in tumors of the adrenal medulla (pheochromocytomas) and due to increased production of adrenaline.
4. Hyperglycemia in hyperthyroidism (see hyperthyroid syndrome, Chapter 73).
5. Hyperglycemia due to insular system B failure.

It is the classic diabetes mellitus due to insufficient insulin secretion (absolute or relative) by the B cells of the islets of Langerhans.

Diabetes mellitus is expressed in the form of two main biological varieties: an immunogenetic one with an absolute insulin defect and another with a relative insulin deficiency due to peripheral insulin resistance, due to disorders of the receptor for this hormone or post-receptor defect for it.

6. Secondary diabetes. In the course of hemochromatosis, pancreatitis and surgical resections of the pancreas.
7. Hyperglycemia in obesity. 60% of highly obese people have carbohydrate intolerance, which is manifested by a slightly abnormal hyperglycemia curve.

The increase in insulin resistance in peripheral fatty tissues is considered a contributing factor to the diabetogenic action of obesity.

Carbohydrate intolerance tends to normalize with weight loss.

8. Hyperglycemia in all forms of physical and psychological stress. They include trauma, infections, hypoxia, hyperthermia, especially in people who have this hereditary trait. The release of catecholamines, which in turn causes glycogenolysis and lipolysis in the liver, frequently reveals prediabetes or latent diabetes.

9. Frequently, a large number of metabolic and endocrine diseases, chronosomal abnormalities and inborn errors of metabolism are accompanied by diabetes.

HYPOGLYCEMIC SYNDROME

CONCEPT

This is the name given to the humoral clinical state that is produced by a homeostatic insufficiency of the glucorretic. gulation by lowering the blood glucose level below 40 mg/dL (or its equivalent 2.2 mmol/L). It can occur in a wide variety of diseases.

SYNDROMOGENESIS OR PHYSIOPATHOLOGY

Maintaining a constant blood glucose level is an essential part of homeostasis.

Physiology teaches us that the concentration of glucose in the blood is the result of two groups of physiological processes:

1. Those that add glucose to the blood: carbohydrate absorption, gluconeogenesis and mobilization of glucose from glycogen (glycogenolysis).
2. Those that remove glucose from the blood: use of glucose by the muscle, adipose tissue, liver, brain, other organs and tissues, according to their needs, hormonal activity, etc.

Human beings feed intermittently, so the intermediate metabolism will be different in the postprandial stage and in the fasting stage.

When the individual ingests food, regulation aims to provide synchronized anabolism and storage of ingested nutrients.

When the levels of circulating energy substances have returned to pre-feeding levels, the fasting state begins. In these moments the regulatory processes come into operation to produce the release of nutrients from the storage sites in an integrated, harmonious and appropriate way to the energy needs of the central nervous system and the periphery.

When the blood glucose level drops below 40 mg/dL in plasma (taken as an arbitrary figure), a reaction of the autonomic nervous system, hormonal changes and disorders in brain function occur that determine the symptoms and signs of hypoglycemia.

From a pathophysiological point of view, hypoglycemia is classified as:

- Hypoglycemia in the postprandial period.
- Hypoglycemia on an empty stomach during the metabolism of endogenous energy substances.

Induced hypoglycemia on an empty stomach.

Hypoglycemia in the postprandial period

It occurs during the digestion of food; It is hypoglycemia due to reaction ("overuse").

A few minutes after eating food it can be shown that the amount of insulin in the peripheral circulation is greater. Although plasma insulin concentration corresponds to increases and decreases in blood sugar concentration, the decrease in insulin level occurs after that of glucose; That is, normoglycemia is established before normoinsulinemia.

On the other hand, insulin reaches the liver directly (portal circulation) before the other tissues and this organ paralyzes its glucose release almost immediately in the postprandial period, only to resume it when blood glucose returns to normal levels.

However, even in normal subjects there may be a delay causing transient hypoglycemia, which is exaggerated in some subjects producing symptoms, precisely in said period.

These disorders express that hepatic production is slow or disturbed and therefore the period of net utilization of glucose from food is prolonged, that is, excessive utilization occurs.

The causes that are most frequently due to this mechanism are:

1. Relative excess of insulin:
   - a) Alimentary hyperinsulinism.
   - b) Hypoglycemia due to reaction, in the case of mild saccharine diabetes that begins during adult life.
   - c) Hypersensitivity to leucine, in infants and children.
2. Accumulation of metabolites that inhibit the reappearance of hepatic glucose production:
   - a) Fructose-1-phosphate aldolase deficiency (hereditary fructose intolerance).
   - b) Galactose-1-phosphate uridinetransferase deficiency (galactosemia).
   - c) Hereditary intolerance to fructose and galactose.
3. Unknown processes:
   - a) Functional hypoglycemia.

Fasting hypoglycemia during the metabolism of endogenous energy substances

It is also called spontaneous hypoglycemia (" defective production")

When you wake up in the morning, all the energy needs of the economy are covered by endogenous resources. At that time, the concentration of sugar in the blood is a consequence of the balance between the continuous use of endogenous glucose and the production of additional glucose by gluconeogenesis from smaller chemical elements, to replace the loss of the substance. The brain depends on glucose for its oxidative needs, which is the main metabolic objective of the fasting state, that is, producing a sufficient amount of glucose to maintain normoglycemia. Since glucose production is carried out at the expense of structural protein catabolism, the second objective is to limit the use of glucose. ization of glucose to the minimum possible, to save protein. The following adaptations facilitate these objectives: the use of glucose is limited in most of the extracerebral tissues, and the most expendable fatty acids are used as oxidant energy; hepatic gluconeogenesis almost completely recovers lactic acid, so that those tissues that depend on glycolysis for energy (for example, erythrocytes, renal medulla) can function without a net expenditure of glucose; and hepatic ketogenesis generates lipids that can cross the blood-brain barrier and replace glucose to some extent in the oxidation needs of the brain, once sufficient ketonemia is established.

Therefore, the net rate of glucose disappearance is the physiological pacemaker of the balance between its utilization and production, which maintains fasting normoglycemia. Hypoglycemia occurs when the liver is unable to adjust to the rate of glucose utilization.

Stuff.

Spontaneous fasting hypoglycemia is a consequence of insufficient hepatic glucose production and can be classified as absolute or relative.

Absolute spontaneous hypoglycemia

Absolute insufficiency of production is rare and occurs when anatomical or intrinsic enzymatic abnormalities in the liver affect its ability to produce a sufficient amount of glucose to counteract the ordinary rates of its utilization in the periphery.

Absolute production failure (due to intrinsic limitations in the liver's ability to produce glucose) is seen in:

1. Diffuse hepatoparenchymal disease.
2. Enzymatic liver defects:
   - a) Final common metabolic pathway:
     - Glucose-6-phosphatase deficiency (glycogen storage disease type I).
   - b) Glycogen replacement:
     - Amylo-l,6-glucosidase deficiency (glycogen storage disease type II).
     - Defects of the phosphorylase enzyme system (glycogen storage disease types VI and VIII).
     - Glycogen synthetase deficiency (aglycogenosis)
   - c) Gluconeogenesis of glucose-6-phosphate from minor chemical elements:
     - Pyruvate carboxylase deficiency.
     - Fructose-1,6-diphosphatase deficiency.

Relative spontaneous hypoglycemia

The relative insufficiency of production is much more frequent. It occurs when normal hepatocytes cannot adjust to normal or supranormal rates of glucose utilization, due to functional limitations of hepatic glucose production, inadequate generation of endogenous substrates for gluconeogenesis, or both processes. In most cases, synergistic factors come into play. For example, direct inhibition of glucose release and acetogenesis in the liver can be combined with limitations in protein lipolysis and catabolism and greater peripheral glucose utilization to produce fasting hypoglycemia due to excess insulin.

Relative failure of production due to insufficient glucose production by (intrinsically normal) hepatocytes is seen in:

1. Excess insulin:
   a) Functional tumors of pancreatic islet cells (Fig. 76.1).
   b) Hyperplasia of pancreatic islet cells (fig. 76.2).
   c) Multiple endocrine adenomatosis I (AEM I syndrome; acronym in English: MEN I).
   d) Neonatal hypoglycemia in children of diabetic mothers.
   e) Neonatal hypoglycemia in cases of erythroblastosis fetalis.
2. Deficiency of counterinsulin hormones:
   a) Panhypopituitarism.
   b) Primary hypoadrenocorticism.
   c) Isolated growth hormone deficiency.
   d) Isolated ACTH deficiency.
   e) Hypothyroidism.
3. Limitations of the availability of substrates to carry out gluconeogenesis:
   a) Ketotic hypoglycemia of childhood.
   b) Fasting hypoglycemia in the terminal phases of pregnancy.
4. Excessive glucose uptake that is not related to insulin:
   a) Very intense exercise.
   b) Renal glycosuria.
5. Unknown processes:
   a) Massive, non-pancreatic tumors that are associated with hypoglycemia.
   b) Idiopathic hypoglycemia in infants and children.

Induced fasting hypoglycemia

It is due to pharmacological interruption of gluconeogenesis, fatty acid oxidation, or both.

Hypoglycemia that occurs spontaneously while fasting must be differentiated from another group of disorders, hypoglycemia induced when fasting.

In the latter case, an exogenous agent has been introduced that can block the endogenous adjustments that maintain normoglycemia when food is not ingested. The resulting hypoglycemia can persist as long as the biological efficacy of the exogenous agent lasts and produce great diagnostic confusion in physicians who are not aware of this possibility. These agents can be:

1. Induced excess insulin:
   a) Insulin administration.
   b) Administration of oral hypoglycemic drugs.
2. Direct inhibition, unrelated to insulin:
   a) Alcoholic hypoglycemia.
   b) Ingestion of unripe ackee fruit (Jamaican vomiting disease).
3. Unknown processes:
   a) Salicylates.

SYNDROMOGRAPHY OR POSITIVE DIAGNOSIS

Clinical picture

When one considers the variety of possibilities and mechanisms of action, both organic and functional, that can alter the homeostatic balance, as well as the influences that disorders of the anterior pituitary, adrenal, pancreas, liver (enzymatic disorders) and the variety of stress of functional origin can exert, it is easy to understand and accept the varied symptoms and signs of hypoglycemic syndrome.

This symptomatology can be permanent or temporary.

Permanent symptoms, when they exist, are almost always manifested by asthenia, fatigue, nervousness and arterial hypotension. The weight is variable; In some, weight loss is the rule, in others, fatness.

The temporary symptoms originate the typical hypoglycemic condition and are characterized because the subject, at certain times, on an empty stomach or in the postprandial period, presents a feeling of hunger, anxiety, sweating, dizziness, indefinite discomfort, lipothymia and

ces comma.

This picture, which is repeated in half of the cases, is not always so simple, but is more complex and depends on the relative level to which the blood glucose dropped, the speed of the drop and the duration of the hypoglycemia.

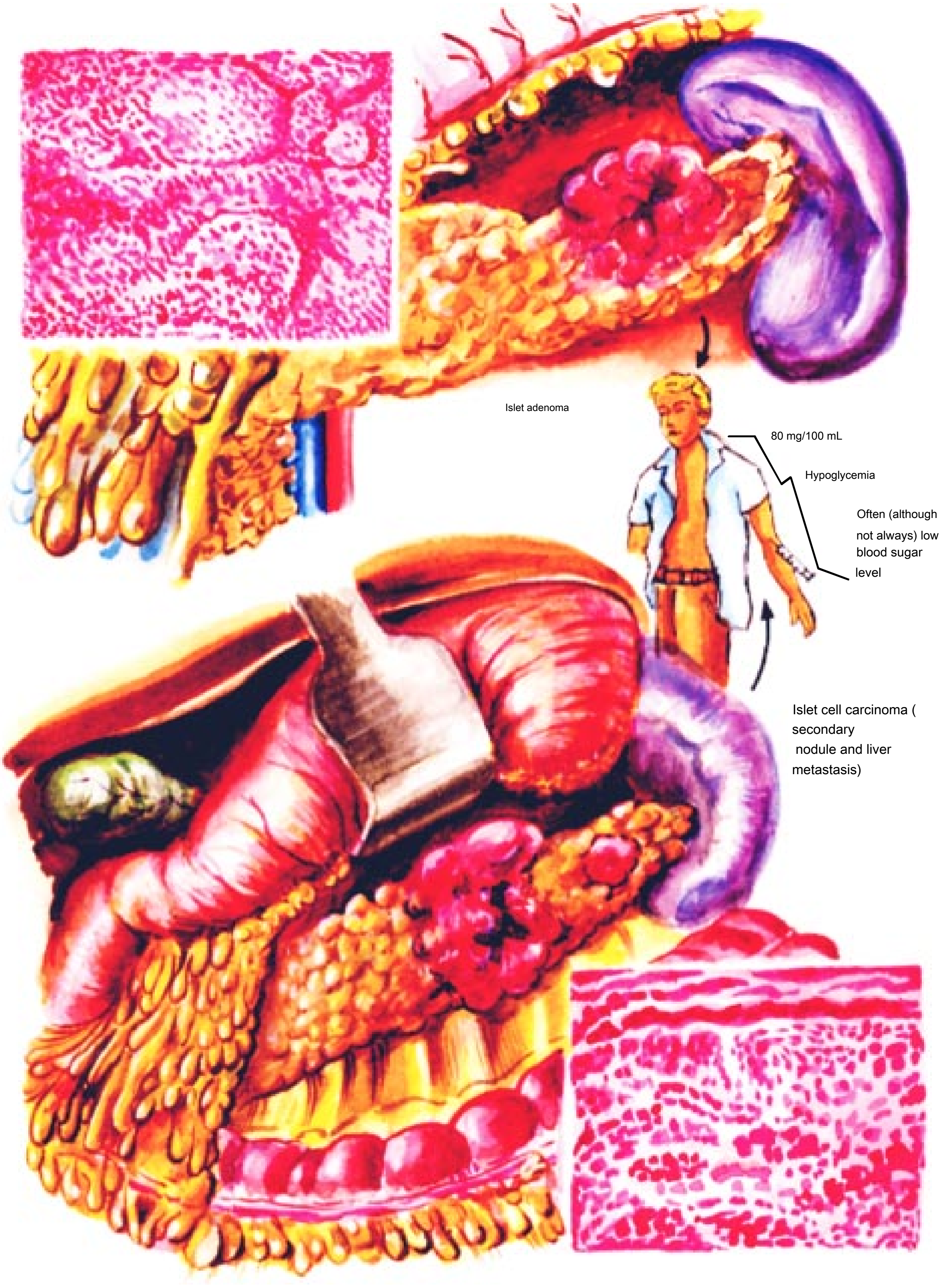

Islet adenoma
80 mg/100 mL
Hypoglycemia
Often (although not always) low blood sugar level
Islet cell carcinoma (secondary nodule and liver metastasis)

Fig. 76.1 Pancreatic B cell tumor.

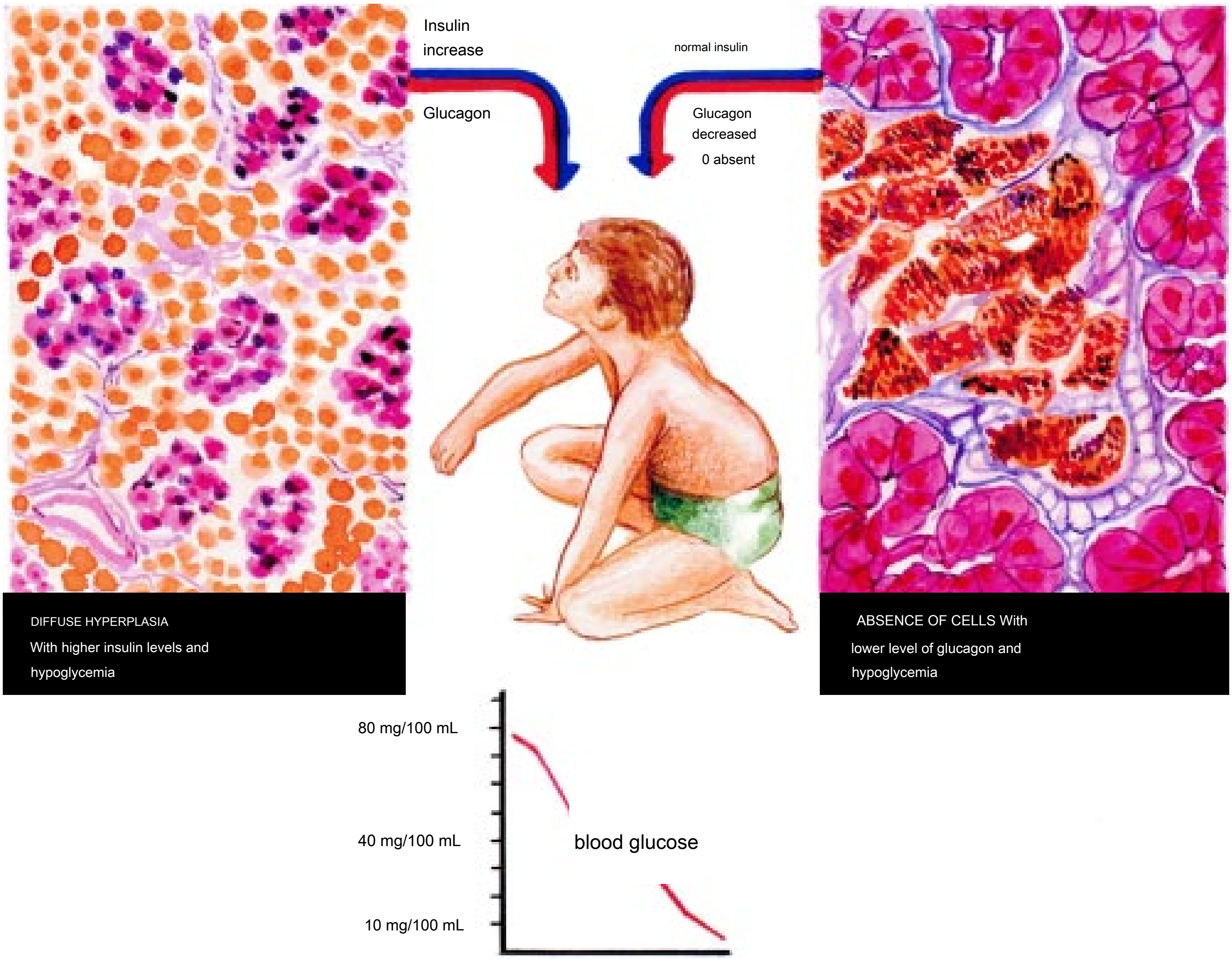

Insulin increase
Glucagon
normal insulin
Glucagon decreased
0 absent
DIFFUSE HYPERPLASIA
With higher insulin levels and hypoglycemia
ABSENCE OF CELLS With lower level of glucagon and hypoglycemia
80 mg/100 mL
40 mg/100 mL
10 mg/100 mL
blood glucose

Fig. 76.2 Pancreatic islet cell dysfunction.

Hypoglycemic crisis

The symptoms of this crisis can be classified into four groups, according to Plattner:

1. Symptoms that are due to the effects of hypoglycemia on the nervous or autonomic system, such as profuse sweating, hunger, abdominal pain that simulates other processes, paleness, palpitations, anginal pain, tremor; The latter are due to the adrenaline reaction that acts in counterregulation.
2. Symptoms that are due to the effects of hypoglycemia on the central nervous system, such as visual disturbances, diplopia, headaches, paresthesias of the lips and fingers, sensation of diffuse luminosity (especially at night).
3. Delayed reaction time (for example, driving a vehicle) and yawning attacks.
4. Psychological symptoms due to hypoglycemia, such as a certain irritability (which often leads patients to categorically refuse sugar or the food offered), depression or, on the contrary, euphoria, decreased ability to concentrate or understand.
5. Symptoms that are due to the effects of hypoglycemia on the muscular system, in particular muscle weakness or fatigue during exercise.

Hypoglycemic coma

When hypoglycemia progresses, the patient loses consciousness and may fall into a more or less profound coma with polymorphous neurological signs: choreoathetotic, clonic or tonic movements; monoplegia or hemiplegia; Tendon areflexia and Babinsky sign. The pupils are dilated, but generally react to light.

Two important elements deserve to be retained for the diagnosis of hypoglycemic coma:

1. The sick person is always very humid and his clothes are soaked with sweat. This finding is not

common in comas of vascular origin and is formally opposed to diabetic coma, in which the patient is more or less dehydrated.

2. Absence of systematization of neurological alterations. For example, hemiplegia with a contralateral Babinsky sign will be observed.

Hypoglycemic coma has a favorable course, although this is not an absolute rule; A serious factor is its long duration (3-4 hours or more), as well as the presence of fever, often linked to cerebral edema.

Complementary exams

When hypoglycemia is suspected, due to the symptoms and signs described above (clinical diagnosis), it must be confirmed by chemical verification of low blood glucose values (humoral diagnosis), as well as the performance of some examinations and dynamic tests that define the etiology of the hypoglycemic syndrome (etiological diagnosis):

1. Dynamic tests. We will only mention them (to know the technique and the results, we recommend consulting a Clinical Laboratory text):
   a) Fasting diet. Fasting is capable of causing intense hypoglycemia in patients with endogenous hyperinsulinism. Tolerance to fasting and negativity of the test rules out organic processes.
   b) 6-h glucose tolerance curve (GTP). It shows us how glycemia behaves at different times of glucose overload.

   The normality of these tests makes it unnecessary to perform the rest of the tests.
2. Liver function tests (to determine liver disease).
3. CT scan of the abdomen to look for neoplasms that hyperproduce insulin: insulinomas.
4. Pituitary and adrenal reserve tests and others that cause hypoglycemia.
5. Electroencephalogram (to differentiate severe hypoglycemia from epilepsy).
6. Electrocardiogram. It shows negative ST segment depression, flattened T wave, and extrasystoles (due to hypokalemia secondary to hypoglycemia).
7. Special tests (only used to specify some rare etiologies): glucagon test, tolbutamide test, determination of galactose, fructose, determination of enzymes, leucine test and others.

ETIOLOGY

The most frequent causes have already been indicated in the pathophysiological classification, so we will not insist on them.

ENDOCRINOMETABOLIC SYSTEM.

GONADAL SYNDROMES

77

MALE HYPOGONADISISM SYNDROMES

The decrease or abolition of androgen hormone secretion (testosterone) and seminiferous sperm secretion is called male hypogonadism.

The causative factors may be:

1. Aplasia or total destruction of the testicles (primitive hypogonadism): castration and eunuchoidism.
2. Lack of its development, secondary to the absence of pituitary gonadotropic stimuli (pituitary hypogonadisms): testicular atrophy.

According to Labhart, the main varieties of hypogonadism are:

- Primary testicular insufficiency.

Secondary testicular insufficiency.

- Hypogonadism of unknown etiology with endocrine alterations.

PRIMARY TESTICULAR INSUFFICIENCY

The testicular gland does not exist or lacks secretory function, or it is partially compromised. The pituitary gland secretes a high amount of stimulating hormones or gonadotrophins (FSH and LH), which is why this type of insufficiency is also called hypergonadotropic hypogonadism. It can take three types, depending on the functions committed:

1. Seminiferous tubular insufficiency. Toxic orchidopathies (X-rays, nitrogen mustards and other cytotoxic agents), orchitis (parotid and melitococcal), bilateral cryptorchidism, germinal aplasia and sclerosing tubular degeneration or Klinefelter's disease.
2. Insufficiency of the interstitial cells of Leydig. Male climacterium.
3. Seminiferous tubular and Leydig cell insufficiency. Castration, congenital anorchidism and total testicular fibrosis.

SECONDARY TESTICULAR INSUFFICIENCY

The cause of the abnormality does not depend directly on the sexual gland, but on a failure of gonadotropic pituitary secretion, which is why it is also called hypogonadotropic or secondary hypogonadism.

It can, like the previous one, be of three types, according to the function committed:

1. Seminiferous tubular insufficiency. Inhibition of gonadotropin secretion due to excess estrogen: non-neutralization (in liver cirrhosis), intense therapy with sex hormones and hyperadrenogenital syndromes.
2. Insufficiency of Leydig interstitial cells. Fertile eunuchs.
3. Seminferous tubular insufficiency of Leydig cells. Isolated hypopituitary gonadotropin failure (idiopathic eunuchoidism and pubertal delays), panhypopituitarisms (Simmonds disease, mental anorexia, cachexia and adiposogenital dystrophy).

HYPOGONADISM OF UNKNOWN ETIOL0GY WITH ENDOCRINE ALTERATIONS

1. Disturbances in spermatogenic maturation.
2. Sterility due to idiopathic hypospermia.

Given the great variety of conditions included in Labhart's classification, we will only briefly present the most characteristic ones: eunuchoidism, Klinefelter syndrome, gynecomastia, male climacteric or andropause, infertility and coeundi impotence.

Eunuchoidism

It is characterized by the absence of seminiferous and interstitial secretion. The lack of the hormone of the genital glands causes different characteristic alterations, depending on whether its absence occurs in childhood or in adulthood. So we have:

- Prepubertal eunuchoidisms.
- Late or post-pubertal geldings.

Prepubertal eunuchoidisms

Syndromography or positive diagnosis (fig. 77.1)

Clinical picture

1. Children's external and internal genital organs.
2. Emptiness of the scrotal contents.
3. Total azoospermia and often aspermia.
4. High-pitched falsetto voice.
5. Absence of hair in the armpits, pubes and face.
6. Trunk with a feminoid configuration: adipose panniculus accumulated in the lower abdomen, breasts and pubis; wide pelvis: scarce muscles.
7. The pubis-plant or pubis-foot distance is greater than 6 cm, in relation to the vertex-pubis distance.
8. The epiphyseal growth lines close late; there is osteoporosis and dorsal kyphosis in 50% of cases; The connecting cartilages undergo delayed ossification.
9. The external habit is the same as that of the adult, but without sex differences or secondary characters; early old age without baldness; white and thin skin; shy, withdrawn character, with a tendency to dissimulation.

Complementary exams

1. FSH and LH increased in blood.
2. Barely detectable testosterone.
3. Human chorionic gonadotropin (HCG) stimulation test: no response, therefore, testosterone is not raised.

Pathophysiology and etiology

1. Surgical castration.
2. Congenital anorchidism.
3. Total fibrosis.

Late or post-pubertal geldings

Syndromography or positive diagnosis

Clinical picture

1. Normal body and skeleton proportions.
2. Regression of secondary sexual characteristics, which is characterized by a decrease in the size of the genitals, as well as the hair on the face, pubis and armpits.
3. Disappearance of erection and libido and appearance of impotence (sometimes, impotence coeundi, that is, impotence for intercourse).

Complementary exams

1. FSH and LH increased in blood.
2. Decreased testosterone in the blood.
3. Response to HCG negative.
4. Biopsy: absence of gonadal parenchyma (atrophy, agenesis and sclerosis).

Pathophysiology and etiology

1. Surgical castration due to malignant tumor or consequences of other interventions: hernias.
2. Tuberculosis.
3. Orchiepididymitis.
4. Castration for medical reasons: parotitis with orchiepididymitis, inflammatory processes (gonorrhea) and luetic processes.

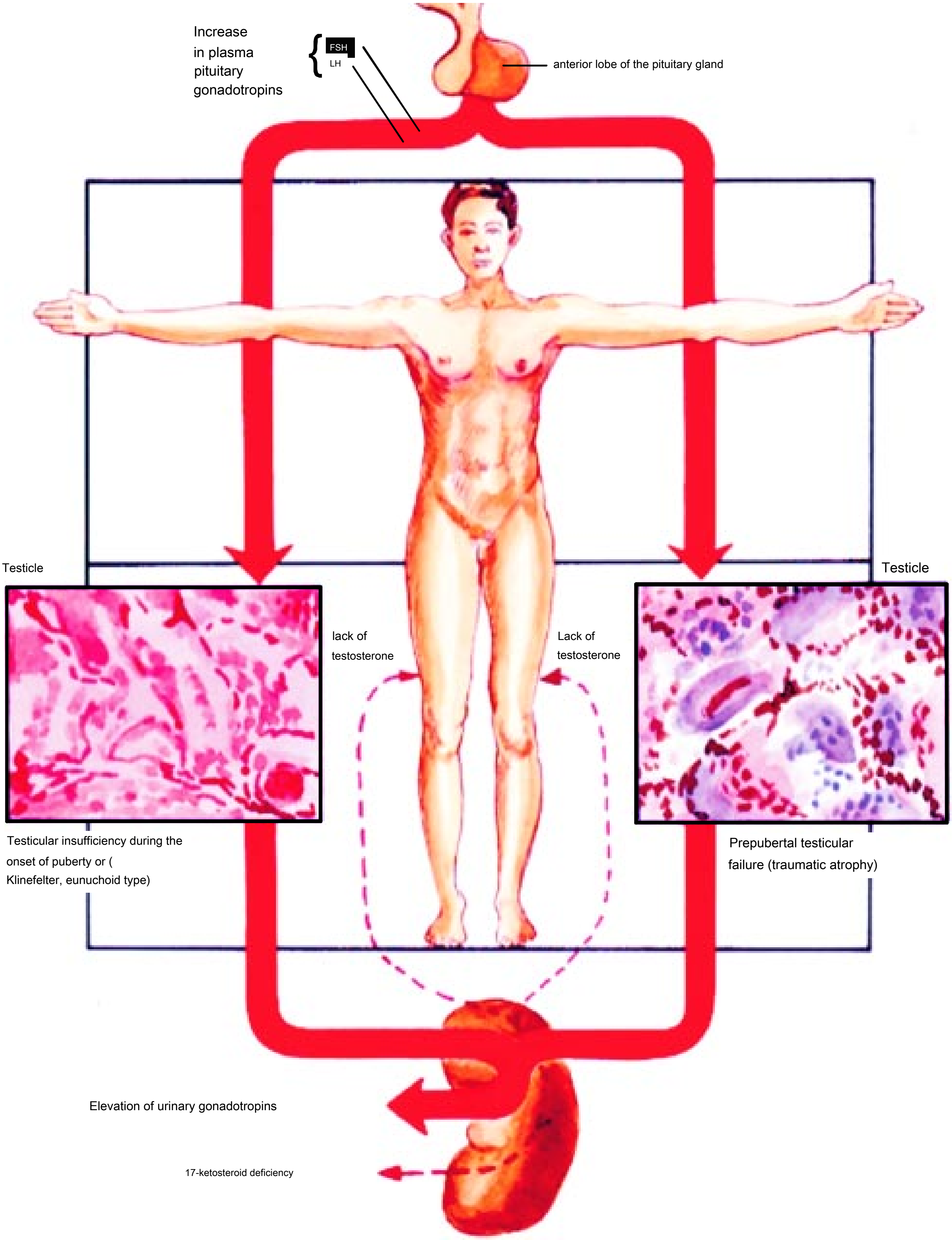

Increase in plasma pituitary gonadotropins
FSH
LH
anterior lobe of the pituitary gland
Testicle
Testicle
lack of testosterone
Lack of testosterone
Testicular insufficiency during the onset of puberty or (Klinefelter, eunuchoid type)
Prepubertal testicular failure (traumatic atrophy)
Elevation of urinary gonadotropins
17-ketosteroid deficiency

Fig. 77.1 Eunuchoidism.

Klinefelter syndrome

It is characterized by the following triad: parenchymal testicular atrophy (primary hypogonadism), postpubertal gynecomastia and increased blood gonadotropins.

In reality, it is an alteration in sexual development, which is manifested by the existence of completely masculine-looking testicles that contrast with the rest of the body's cells, which can be chromatin-negative or chromatin-positive.

Syndromography or positive diagnosis (fig. 77.2)

Clinical picture

Patients grow and develop well, testosterone reaches variable levels in the blood, therefore, secondary sexual characteristics exist: hair on the pubes, face, conservation of libido and sexual impulses.

However, the damage to the spermatogenic epithelium is irreparable and there is azoospermia and sterility.

Psychological sex turns out to be, generally, neutral OR masculine.

Complementary exams

1. FSH and LH elevated and sometimes normal.
2. Decreased and sometimes normal testosterone.
3. Chromatin in oral mucosa is positive (Barr's corpuscle) (female type). The karyotype is 47,XXY.

Pathophysiology and etiology

The XXY chromosome formula (genotype) usually depends on the meiotic nondisjunction in the maternal gamete, with which the egg has two X chromosomes.

Meiotic nondisjunction during spermatogenesis has also been corroborated.

A relationship has been observed with the age found in the parents. It has been seen that this syndrome is more frequent when pregnancy occurs later than at the usual age.

At birth, the child generally does not present obvious clinical stigmata, but upon puberty, a series of pathophysiological mechanisms occur, such as:

1. Increase in pituitary gonadotropins (FSH and LH), which explains gynecomastia.
2. Progressive hyalinization and testicular fibrosis, so the testicles appear small and hard.
3. Obliteration of the seminiferous tubules, causing azoospermia, with abnormal Leydig cells (hyperplasia of the same), but with variable secretion of testosterone. This causes a virilization pattern that shows a clinical spectrum, from almost normal to poor.
4. It is known that the testicles of these patients are dysgenetic.

Gynecomastia (fig. 77.3)

It is frequently a sign of hypogonadism. When it appears during puberty, it worries many adolescents. Sometimes it is hereditary and evolves without psychosexual disorders or gonadal anomalies. However, young people who suffer from it live uncomfortable and withdrawn, avoiding going to beaches, gyms, etc.

Syndromogenesis or pathophysiology

Apart from pubertal and idiopathic, which are the most common, gynecomastia can appear in the following processes:

1. Endocrine:
   a) Testicular insufficiency (hypogonadism): castration, eunuchoidism and Klinefelter syndrome.
   b) Excess estrogen: chorionic testicular tumors (teratomas), feminizing tumors of the adrenal cortex, FSH and LH hyperfunctioning tumors.
   c) Deficient neutralization of estrogens in the liver due to liver failure.
2. Iatrogenic:
   a) Application of estrogens in prostate cancers.
   b) Excessive hormonal treatments (estrogens, gonadotropins, testosterone, excessive DOCA and corticosteroids).
   c) Prolonged use of medications with phenanthrenic nuclei: vitamin D and digitalis; other medications (cimetidine, spironolactones, etc.).
3. Other processes:
   a) Leprosy.
   b) Leukemia.
   c) Bronchial cancer.
   d) Spinal cord injuries: paraplegia, intercostal nerve sections, among others.

Malnutrition or interference with the neutralization of estrogens by the liver is involved in all of them.

Male climacterium or andropause

Physiological involution of men that generally occurs between 50 and 60 years of age.

Syndromography or positive diagnosis

Clinical picture

The symptoms are similar to those of female menopause: nervousness, sadness (melancholy), loss of memory and interest, insomnia, decreased libido, but not genetic power, impotence and tendency to prostate hypertrophy.

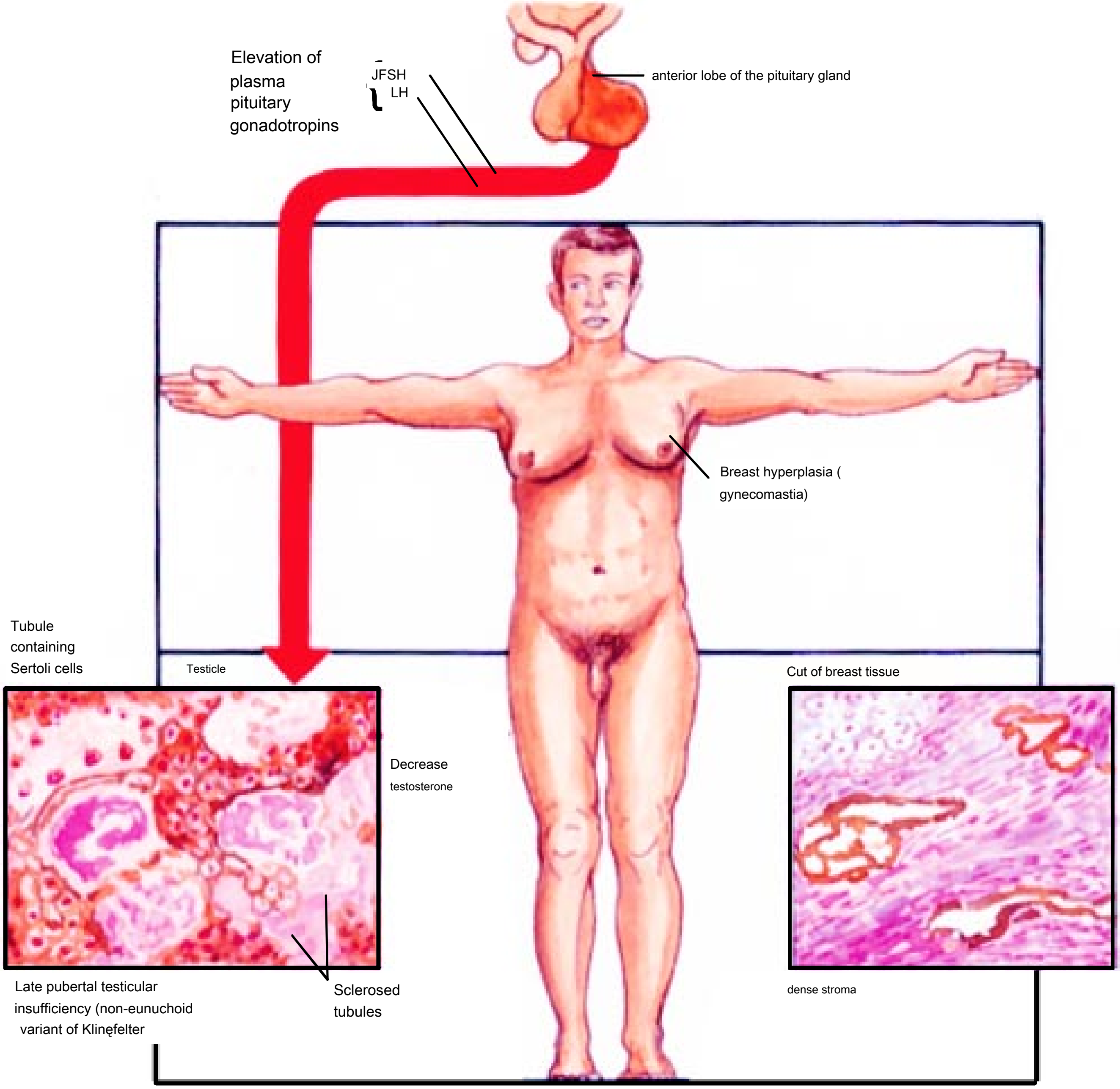

Elevation of plasma pituitary gonadotropins
JFSH
LH
anterior lobe of the pituitary gland
Breast hyperplasia (gynecomastia)
Tubule containing Sertoli cells
Testicle
Cut of breast tissue
Decrease testosterone
Late pubertal testicular insufficiency (non-eunuchoid variant of Klinefelter
Sclerosed tubules
dense stroma
Typical: 47,XXY
Generally positive Baar corpuscles
(female type: generally the chromosome formula is 47,XXY; but cases 48,XXXY; 49, XXXXY; 48,XXYY;
49,XXXYY and cases of mosaicism

Fig. 77.2 Klinefelter syndrome.

The diagnosis is difficult to establish; It requires ruling out other conditions with similar symptoms: depressive neurosis, incipient arteriosclerosis and arterial hypertension.

Complementary exams

1. Total testosterone is normal, but free or unconjugated testosterone is decreased.
2. FSH and LH, within normal or slightly high limits.

Infertility and impotence coeundi

Marriages are 10% sterile. Of these, 40-50% of cases are due to male sterility.

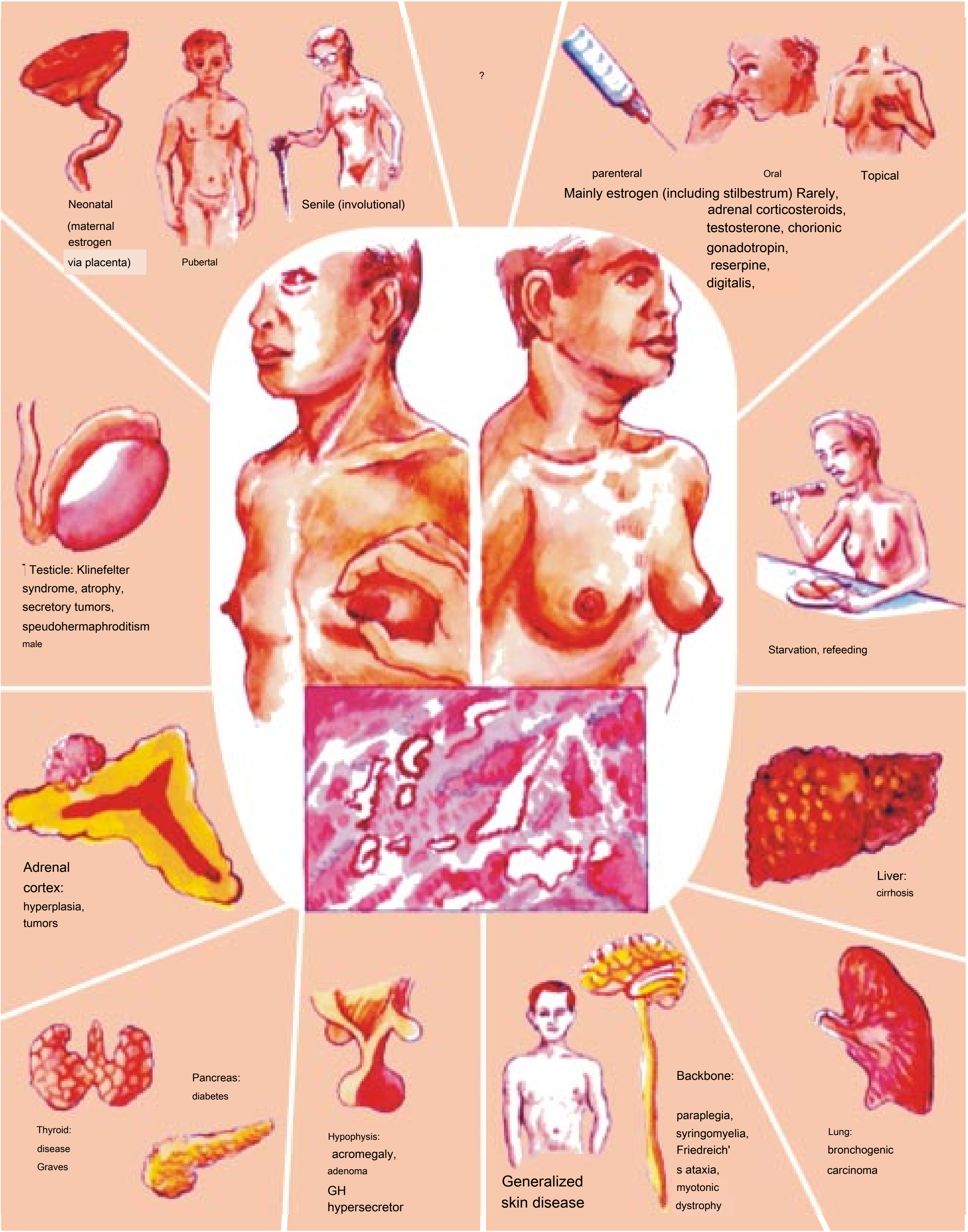

Neonatal
(maternal
estrogen
via placenta)
Pubertal
Senile (involutional)
?
parenteral
Oral
Topical
Mainly estrogen (including stilbestrum) Rarely,
adrenal corticosteroids,
testosterone, chorionic
gonadotropin,
reserpine,
digitalis,
Testicle: Klinefelter
syndrome, atrophy,
secretory tumors,
speudohermaphroditism
male
Starvation, refeeding
Adrenal
cortex:
hyperplasia,
tumors
Liver:
cirrhosis
Pancreas:
diabetes
Thyroid:
disease
Graves
Hypophysis:
acromegaly,
adenoma
GH
hypersecretor
Generalized
skin disease
Backbone:
paraplegia,
syringomyelia,
Friedreich'
s ataxia,
myotonic
dystrophy
Lung:
bronchogenic
carcinoma

Fig. 77.3 Gynecomastia.

If the testicles are of normal size, the most common cause is usually:

1. Postorchitic occlusions of the epididymal or ejaculatory ducts, in which surgical correction is possible.

2. Impotence coeundi, which may depend on:
   a) Anatomical defects: enormous hernias, balanophimosis, penile
   b) Endocrine disorders: diabetes mellitus, hypothyroidism, hyperthyroidism, Addison's disease others.
   c) Neurological or cardiovascular disorders: myelopathies, arteriosclerosis, senility.
   d) If the cause of impotence coeundi is not one of the three mentioned, then it will be psychogenic and due to emotional conflicts, such as depressive neuroses and neurasthenia.

FEMALE HYPOGONADISM SYNDROMES

Below we point out the most frequent cases of female hypogonadism:

Castration.

- Menopause.

Turner syndrome.

Noonan syndrome.

Polycystic ovary syndrome (Stein-Leven-thal).

CASTRATION

It generally occurs at the woman's full sexual maturity, due to the surgical removal of the ovaries due to diseases of the adnexa (artificial climacterium).

Syndromography or positive diagnosis

Clinical picture

The symptoms appear abruptly in acute form and are characterized by:

1. General symptoms. Flashes of heat, chills, fainting, acroparesthesias, vague pains throughout the body.

2. Psychological disorders. Feeling of anguish, sadness, nervousness and insomnia, headaches.
3. Signs of arthritis. In knees and shoulders, mainly.

4. High blood pressure and obesity.
5. Genital symptoms. Cessation of menstruation, atrophy of the uterus and vagina, vulvar pruritus.

Complementary exams

1. Increased FSH and LH.
2. Barely detectable testosterone.

Pathophysiology and etiology

It is almost always surgical, by removing the ovaries.

rivers.

MENOPAUSE

It is produced by the physiological involution of sexual function; It occurs between 45 and 55 years of age. It is sometimes preceded by a premenopausal period, in which estrogenic predominance causes menstrual disorders with metrorrhagia.

Syndromography or positive diagnosis

Clinical picture

In 75% of cases it evolves with pathological symptoms, similar to those of castration:

1. General symptoms. Waves or flashes of heat, profuse sweating, migraine headaches, frequent and urgent urination and increased blood pressure.
2. Arthritic disorders. Cervical dysarthrosis or scapulohumeral periostitis with painful, nocturnal brachial paresthesias; crunchy knees.
3. Marked variations in secondary sexual characteristics. Weight gain, the silhouette is deformed, sagging and hanging breasts, increased hairiness.
4. Genital disorders. Decreased volume of the ovaries and uterus, vaginal cornification, vulvar craurosis.

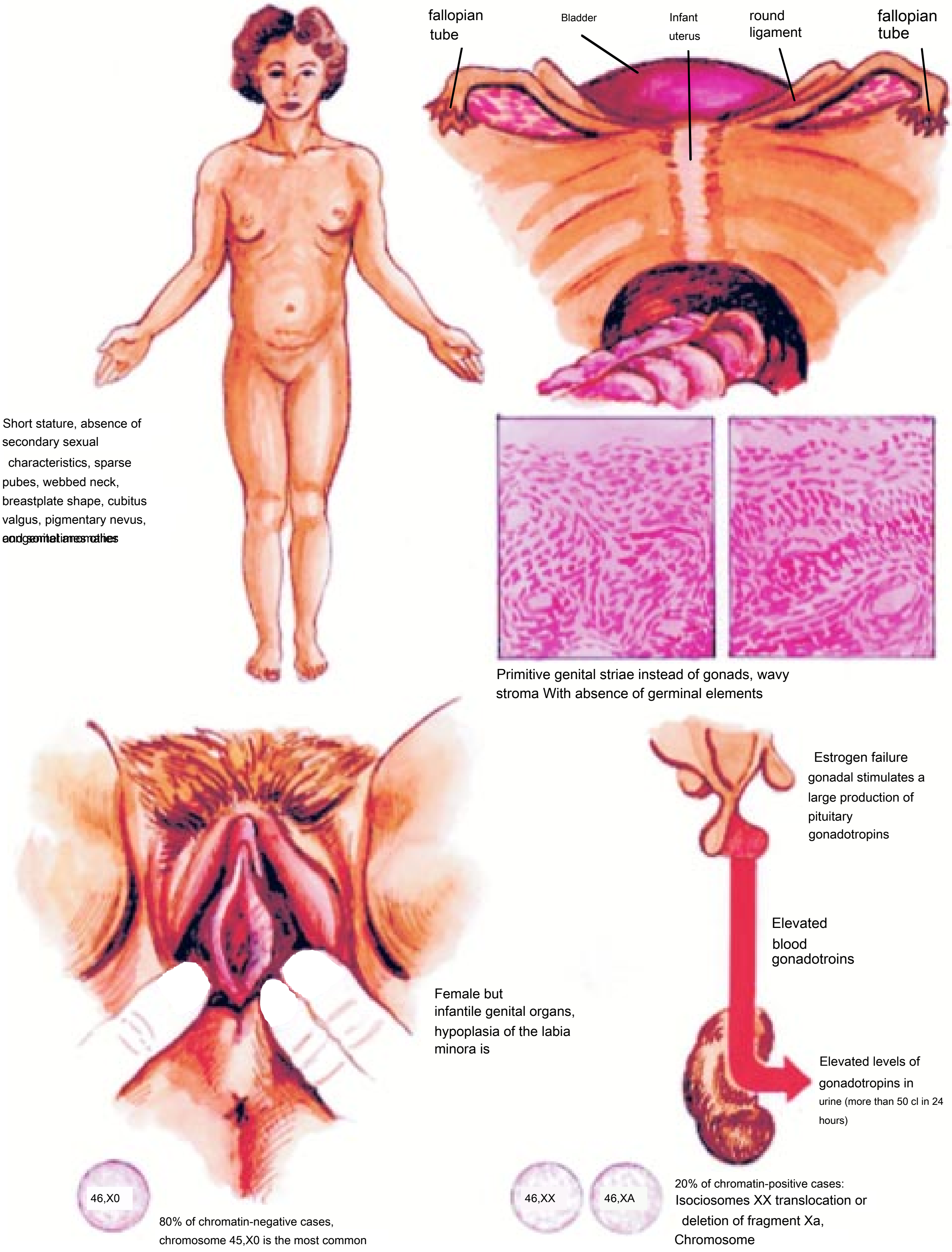
5. Libido and orgasm. They can be preserved even for years.
6. Psychogenic disorders. Sometimes melancholy, sadness, desire to cry; other times restlessness, anxiety, exaggerated eroticism; and on other occasions, serenity and judgment are enriched, gaining a stability that they did not possess.

Complementary exams

1. Endometrium with glandulocystic hyperplasia.
2. Hormonal dosages demonstrate:
   a) FSH and LH increased.
   b) Gonadotropins sometimes increased.
   c) Decreased estrogens (17-B-estradiol).

TURNER SYNDROME

Also called Turnerian gonadal dysgenesis, it constitutes a congenital anomaly with multiple malformations, which is observed in the female sex (fig. 77.4).



Short stature, absence of secondary sexual characteristics, sparse pubes, webbed neck, breastplate shape, cubitus valgus, pigmentary nevus, and sometimes other congenital anomalies

Primitive genital striae instead of gonads, wavy stroma With absence of germinal elements

Estrogen failure gonadal stimulates a large production of pituitary gonadotropins

Elevated blood gonadotroins

Female but infantile genital organs, hypoplasia of the labia minora is

Elevated levels of gonadotropins in urine (more than 50 cl in 24 hours)

80% of chromatin-negative cases, chromosome 45,X0 is the most common

20% of chromatin-positive cases: Isociosomes XX translocation or deletion of fragment Xa, Chromosome

Fig. 77.4 Turner syndrome.

Pathophysiology and etiology

The chromosome formula of these patients is 45.X0.

It is the absence of a second sex chromosome (X chromosome monosomy). This anomaly is due to an abnormal second sex chromosome, an absence that may be a consequence of a disjunction or loss of chromosomes during gametogenesis in either parent, resulting in a sperm or egg lacking any sex chromosome. It is a disorder due to mitotic nondisjunction. This chromosomal alteration determines:

1. Increase in FSH and LH in blood and urine.
2. Absence of estrogens in plasma and urine.
3. Presence of fibrous bands that replace normal ovaries (streak gonad).

Syndromography or positive diagnosis

Clinical picture

There are four characteristics that distinguish this syndrome: ovarian agenesis, pterygium colli (fin-shaped extensions of the neck folds), low hair set and cubitus valgus.

Other events are generally associated, such as short stature, due to lack of cartilaginous response to the somatotropic hormone; slightly marked axillopubic hair, due to adrenal androgen stimulation, and broad chest, due to delayed closure of the costal cartilages.

It is not uncommon for this syndrome to be accompanied by other congenital anomalies; for example: pigmentary nevus, epicanthus, lax cutis, aortic coarctation, hypoplastic breasts, syndactyly and deaf-muteness.

Complementary exams

1. LH and FSH levels are high.
2. Laparoscopy demonstrates uterovarian absence with an outline in the form of cords or fibrous bands (streak gonad).
3. A negative oral chromatin as opposed to that of the normal female with 45,X0 karyotype.
4. Estrogens in blood and urine not detectable.

NOONAN SYNDROME

It can appear in both sexes. It has the sexual and somatic characteristics of Turner syndrome, but has a normal chromosome formula, both in females and males (46.XX and 46. XY).

This last variant was previously called male Turner syndrome. In this case, to the sexual and somatic characteristics of the Turner, are added the following signs: cryptorchidism, testicular hypoplasia and infantilism.

In all cases the result is always decreased libido and infantile feminine or masculine psyche with feminoid tendency.

Syndromogenesis or pathophysiology

The gonads of these patients may present different degrees of differentiation, but they are present, although they are dysgenetic and can cause:

1. Somaic anomalies similar to Turner syndrome.
2. Genetic short height or stature.
3. FSH and LH increased or normal at the lower limit of normal.
4. Decreased or normal testosterone in the lower limit in the 46.XY variety.
5. Low or normal estrogens in the 46,XX variety.

POLYCYSTIC OVARY SYNDROME (STEIN-LEVENTHAL)

It begins between 18 and 25 years of age and is characterized by amenorrhea, sterility, hirsutism and occasional obesity accompanied by an increase in the size of both ovaries, which show a polycystic appearance (Fig. 77.5).

Syndromogenesis or pathophysiology

It is estimated that this syndrome depends on high levels of LH, but without causing ovulation, which determines a disorder in the hypothalamicpituitary axis with anatomical alterations in the ovaries with multiple atretic follicles on the thickened ovarian surface.

ment of the theca interna with bilateral enlargement of both ovaries, which causes infertility, hirsutism, amenorrhea and poor virilization.

It is estimated that this syndrome depends on an excess of pituitary luteinizing hormone (LH) and progesterone.

Syndromography or positive diagnosis

Clinical picture

1. Menstrual disorders: secondary amenorrhea, sometimes oligomenorrhea with interspersed menorrhagia.
2. Anovulatory cycle: sterility.
3. Normal-looking breasts.
4. Obesity in 10% of cases.
5. Hirsutism.
6. Virilism: clitoral hypertrophy, thick voice and acne.
7. On palpation: large polycystic ovaries.

Complementary exams

1. Hormonal tests:
   a) Elevated LH and normal FSH in the blood.
   b) Normal or occasionally elevated plasma estrogens.
   c) Elevated urinary pregnandiol.
2. Imaging examinations:
   a) Gynecological ultrasound: increase in size of both ovaries with multiple cysts in the cortex.
   b) Pelvineumography: allows visualization of the ovaries, which have a smooth surface and are almost double the normal size.
   c) Laparoscopy: increase in size of both ovaries with an irregular, pearly white surface with multiple atretic cysts below the cortex and thickening of the theca interna.

INTERSEXUALITY AND HERMAPHRODITISM

They are due to the coexistence of somatic characteristics of both sexes in the same individual.

SYNDROMOGRAPHY OR POSITIVE DIAGNOSIS

Clinical picture

The external genitalia are in opposition to the histological characters of their gonads or to the cellular chromosomal sex.

Almost all cases are congenital and infantile; The most common form is male pseudohermaphroditism, which is characterized by having female external genitalia and male gonads and cellular chromosomal characteristics.

Genuine or true hermaphroditism is extremely rare and is characterized by the coexistence of male and female gonads in the same patient.

Complementary exams

Laparoscopy or laparotomy shows grayish white ovaries with intraovarian follicular cystic degenerations varying in size between a grain of rice and a pea.

HYPERGONADISM SYNDROMES

MALE HYPERGONADISM. MALE HYPERGENITALISM

It is a syndrome that depends on the hyperproduction of testosterone. Its clinical and symptomatic manifestations They are not well defined and consist of: excessive development of the testicles, rather short stature, wide and reddish facies, short neck and increased libido that encourages sexual excesses.

The differential diagnosis should be made with psychogenic

FEMALE HYPERGONADISM0

The most common female hypergonadisms are:

-Hyperfolliculinism or hyperstrinism.

- Hyperluteism.

Hyperfolliculinism or hyperstrinism

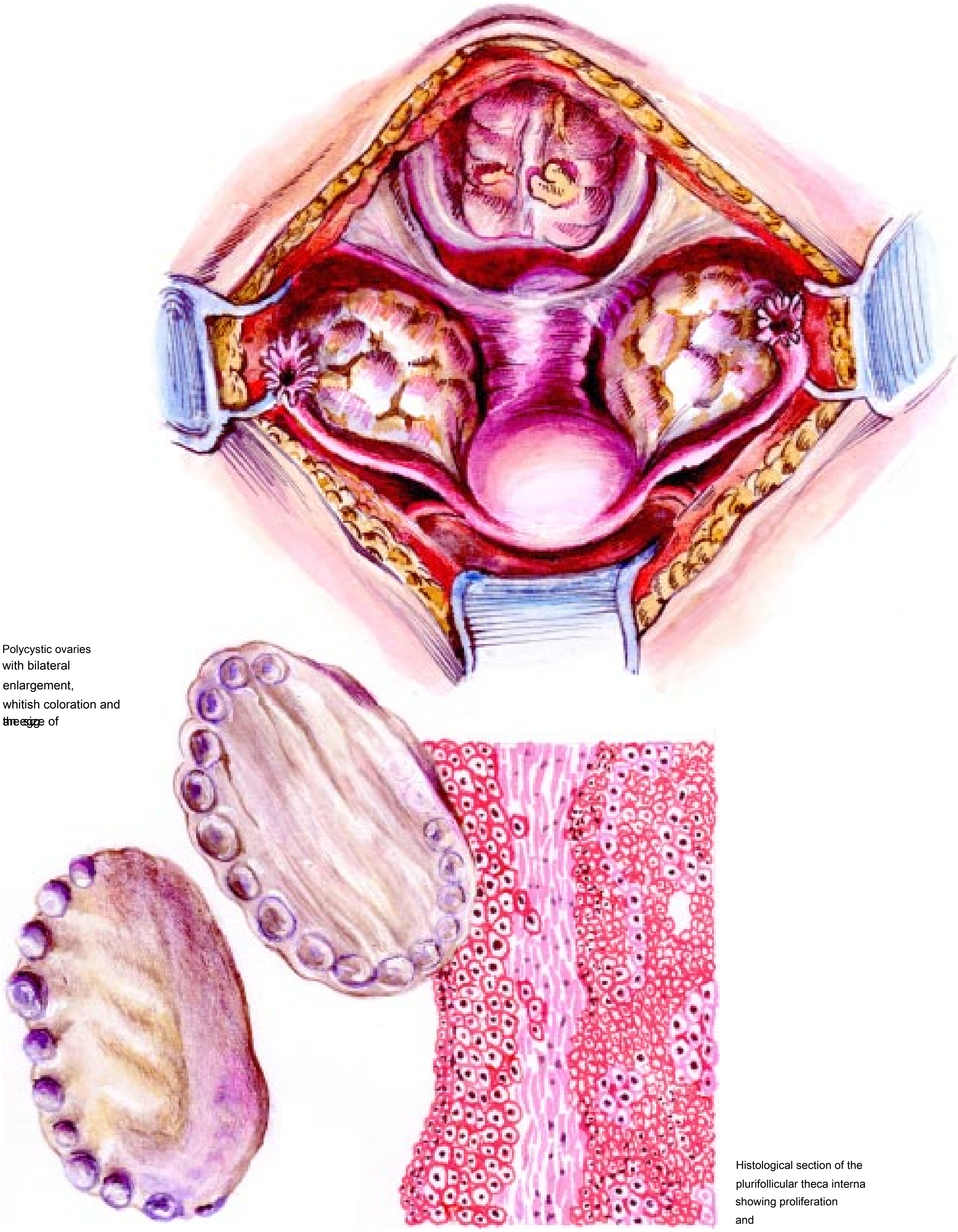
This syndrome results from the predominance of follicular hormone over progesterone. It is characterized by the so-called premenstrual syndrome, which consists of:

1. Hydrosaline retention, which produces painful breast congestion, called mastodynia, with cellulite and cystic mastopathy, concomitant abdominal bulging, general swelling with a sensation of numbness and hoarse voice.
2. Cortical and hypothalamic nervous hypersensitivity, characterized by migraine, increased thirst (polydipsia), excessive appetite (polyphagia), insomnia, clumsiness and character variations (irritability, restlessness, fatigue), dyspepsia and acrocyanosis.
3. Allergic disorders. They are manifested by urticaria, Quincke's edema and premenstrual asthma.
4. Increased blood pressure.
5. Relaxation of the ligaments, due to hyperfolliculinemia, which causes easy twisting of the ankles.
6. The menstrual cycle tends to be hypermenorrheic and polymenorrheic, sometimes normal or oligomenorrheic.
7. In adolescence there may be dysmenorrhea and sterility.
8. During the physical examination, increased breast development and external genitalia (large lips, etc.) can be seen.

Complementary exams

Complementary tests in hyperfolliculinism or hyperstrinism are:

1. Increased gonadotropins.
2. Hyperestrogenia.
3. Increased phenolsteroids.
4. Pregnandiol decreased.
5. Vaginal smear with increased eosinophilic cells.



Polycystic ovaries
with bilateral
enlargement,
whitish coloration and
the size of an egg

Histological section of the
plurifollicular theca interna
showing proliferation
and

Fig. 77.5 Polycystic ovary syndrome (Stein-Leventhal).

Pathophysiology and etiology

Hyperstrinism can be:

1. Absolute:
   - a) Due to hyperproduction of folliculin: ovarian tumors (thecal cells and granulosa cells), adrenal tumor, excess of pituitary folliculostimulin and iatrogenic due to therapeutic excesses with estradiol.
   - b) Due to poor destruction of folliculin in the liver: liver cirrhosis.
2. Relative:
   - a) Pituitary ovarian imbalances, especially in: premenopause, menopause and after abortions OR childbirth.
   - b) Hyperexcitability of the cerebral cortex or hypothalamus.

Hyperluteism

It appears due to excess progesterone. In primary luteism (due to ovarian hyperfunction) the most characteristic sign is menstrual delay with pseudo-gravid phenomena: breast turgor and pigmentation of the areola.

After the age of 40, deciduform metritis usually appears, with abundant menses. The diagnosis is difficult; Sometimes, the condition corresponds to a placental tumor: hydatidiform mole or chorioepithelioma.

Complementary exams

The complementary exams in primary luteism are:

1. Increased urinary pregnadiol.
2. Persistence of the corpus luteum.
3. Vaginal smear with few eosinophilic cells.

In luteism due to placental tumor we find hyperprolactinemia, more than 60,000 U.

INDEX OF VOLUME 2

Section II CLINICAL PROPEDEUTICS (Continued)

50 Nervous system. Clinical history and alterations in the general examination / 709

Medical history /709
- Interrogation / 709
- Physical exam /711
- Instrumental exams / 711

Alterations in the general physical examination /711
- Pathological facies / 711
  - Semiography /711
    - Facies of the patient with capsular hemiplegia during coma / 711
    - Pseudobulbar patient facies / 711
    - Facies of Parkinson's disease and parkinsonian syndromes / 712
    - Facies of myasthenia gravis/712
    - Tetanic facies (or sardonic laughter) and strychnine poisoning / 712
- Standing attitude /713
  - Semiography/713
    - Parkinson / 713
    - Capsular hemiplegia with spasticity / 713 Advanced ataxia / 713
    - Myopathies or muscular dystrophies/714
    - Spastic paraplegia of the extension type/ 714
    - Korea / 714
- Attitude in bed/714
  - Semiography /714
    - Hemiplegia during coma / 714
    - Opisthotonos, emprostotonos, pleurototonos / 714
    - Orthotones / 714
    - Shotgun trigger attitude / 714 March / 714
  - Semiography and semidiagnosis/ 714
    - Paretic gait / 715
    - Spastic gait / 715
    - Ataxic gait / 715
    - Walking with small steps /716
    - Digitized walking/ 716
    - March of primary myopathies / 716 March of clawn in Korea / 717
    - Toad march / 717

51 Nervous systemSO. Alterations in your private physical examination /718

Trophism / 718
- Concept / 718
- Semiogenesis or physiopathology / 718
- Semiotechnics /719
- Semiography and semidiagnosis /719
  - Classification
    - Skin and nail disorders / 719
    - Joint disorders / 719
    - Bone atrophy or osteoporosis / 719
    - Semiological alterations typical of myotrophism 7719

Reflectivity alterations / 720
- Semiography and semidiagnosis, / 720
  - Normal reflexes / 721
  - Hyporeflexia and areflexia / 721
    - Osteotendinous or deep areflexia / 721
    - Superficial or cutaneous-mucous areflexia / 722
  - Hypopereflexia /722
    - Positive plantarcutaneous reflex or Babinski sign/ 722
    - Clonus/723

Alterations in muscle tone / 723
- Semiography and semidiagnosis / 723
  - Hypertonia /723
  - Hypotonia / 724
  - Dystonia/ 724

Alterations of motility/ 724
- Semiography and semidiagnosis /724
  - Paralysis due to central motor neuron injury and peripheral motor neuron injury / 724
- Particular study of hemiplegia: Hemiplegic syndrome/724
  - Beginning Forms / 724
  - Recognition of hemiplegia during coma / 724 Characteristics of hemiplegia outside of coma / 725

Flaccid hemiplegia/725
Spastic hemiplegia /726
Larvate hemiplegia /726
Classification of hemiplegia /726
Topographic diagnosis of hemiplegia (diagnosis of the height of the lesion) / 726
Cortical hemiplegia /726
Subcortical hemiplegia /727
Capsular hemiplegia / 727
Thalamic hemiplegia / 727
Truncular and bulbar pyramidal syndromes / 727 Ataxia / 728
Alterations of static coordination / 729
Semiological importance of Romberg's sign / 729
Alterations of dynamic coordination / 729
Semiography and semidiagnosis / 729
Praxia alterations / 730
Definitions of praxia / 730
Rating / 731
Semiogenesis / 731
Semiotechnics / 733
Semiography/ 733
Semiodiagnosis / 734
Sensitivity alterations / 734
Semiography and semidiagnosis/ 734
Sensitivity dissociation syndromes / 734
Spinal sensory syndromes / 734 Radicular sensory syndromes 737
Cauda equina syndrome / 738 Peripheral sensory syndromes / 738

52 Nervous system. Pair alterations
cranials / 739

Olfactory nerve: I pair/ 739
Semiography /739
Semiodiagnosis / 740
Optic nerve: II pair / 740
Semiography /
740 Semiodiagnosis / 741
Injuries to the visual apparatus /741
Visual defects / 741
Oculomotor, trochlear, and motor nerves
external eyepiece (abducens): III, IVy VI pairs /741
Semiography and semidiagnosis /741
Nuclear ophthalmoplegia / 742
Infranuclear ophthalmoplegias / 742
Supranuclear ophthalmoplegias / 742
Semiography and special semidiagnosis of the portion intrinsic of the III pair / 742
Pupillary morphological alterations / 742
Alterations in pupillary reflexes / 743
Trigeminal nerve: V pair /744
Semiography and semidiagnosis / 744
Facial nerve: VII nerve /744
Semiography and semidiagnosis / 744 Central facial paralysis / 744
Peripheral facial paralysis / 744
Semiographic alterations of the sensory portion / 745
Statoacoustic nerve: VIII pair / 746 Cochlear nerve / 746
Semiography and semidiagnosis / 746
Vestibular nerve or branch / 746
Semiogenesis and semiography / 746
Semiodiagnosis / 746
Glossopharyngeal, pneumogastric (vagus) and accessory nerves: IX, X and XI pairs / 747
Glossopharyngeal nerve: IX pair / 747
Semiography / 747
Semiodiagnosis / 747
Pneumogastric (vagus) nerve: X pair / 747
Semiography and semidiagnosis /747
Accessory nerve: XI pair / 748
Semiography and semidiagnosis / 748
Greater hypoglossal nerve: XII pair / 748
Semiography/748
Semiodiagnosis / 748

53 Nervous system. Additional investigations / 749 Lumbar puncture and study of cerebrospinal fluid (CSF) or cerebrospinal fluid / 749
Anatomy of the system that houses the LCR/ 750
LCR/750 Features
Lumbar puncture technique / 750
Queckenstedt-Stookey test/751
CSF examination/ 752
Normal characters / 752
Amount of CSF required for each test / 752

Indications for lumbar puncture /752
Contraindications of lumbar puncture / 752
Information you can provide / 752
Imaging examinations / 752 Simple skull x-ray / 753 Indications / 753

Information you can provide / 753
Pneumoencephalogram / 753
Indications / 753
Information you can provide / 753 Risks/ 753
Cerebral angiography / 753
Technique / 753
Information you can provide / 754
Risks / 754
Myelography / 754
Echoencephalography or brain ultrasound / 754
Computed axial tomography (CAT) / 754
Information provided / 755
Magnetic resonance imaging (MRI) / 755
Exploration of the central nervous system using radioisotopes / 756
Brain scan /756
Positron emission tomography (PET)/ 756 Photon emission tomography (SPECT) / 756
Electroencephalography / 756
Normal electroencephalogram / 757
Abnormal electroencephalogram / 757

Directions / 758
Evoked potentials / 758
Directions / 758
Information they can provide / 758
Electromyography and electrical stimulation of muscles and peripheral nerves / 758
Directions / 759
Biopsies / 759
Neuromuscular biopsy / 759

54 Main syndromes of the nervous system / 760

Motor syndromes / 760
Paraplegic syndrome / 760
Concept / 760
Flaccid paraplegia/ 761
Syndromogenesis or pathophysiology / 761
Syndromography or positive diagnosis / 761
Etiology / 761
Spastic paraplegia / 761
Syndromogenesis or pathophysiology / 761
Syndromography or positive diagnosis / 762
Etiology/ 762
Flascospastic paraplegia / 762
Etiology/ 762
Extrapyramidal syndrome (involuntary movements) / 762
Anatomophysiological count/762
Abnormal movements or hyperkinesias / 764
Tremors / 765
Myoclonus / 766
Choreic movements / 767
Dystonokinesias / 767
Muscle spasms / 768
Tics /768
Hemiballism /768
Fibrillations and fasciculations / 769
Seizures / 769
Psychogenic hyperkinesias / 769
Parkinsonian syndrome / 769
Concept/ 769
Syndromogenesis or pathophysiology / 769
Syndromography or positive diagnosis / 770
Etiology / 771
Convulsive syndrome / 771
Concept / 771
Syndromogenesis or pathophysiology / 771
Syndromography or positive diagnosis / 772
Generalized tonic-clonic or grand mal seizure /772
Partial seizure, focal motor, Jacksonian or Bravais-Jackson / 773
Status epilepticus / 773
Tonic crisis /773
Complementary exams / 773
Etiology / 774
Sensory syndromes / 774
Headache / 774
Concept / 774
Syndromogenesis or pathophysiology / 774
Rating / 774
Syndromography and etiology / 774
Migraine or migraine / 775
Histamine headache (cumulative headache, Horton syndrome) / 775
Headache due to mental tension / 775
Headache as a symptom of brain tumor/775
Trigeminal neuralgia / 775
Other causes of headaches / 775
Brain stem syndromes / 775
Peduncular syndromes /777
Peduncular alternating hemiplegia (Weber syndrome) / 777
Superior Foville or peduncular Foville syndrome /777
Benedikt or posterior peduncular syndrome / 778 Pontine syndromes / 778
Anterior pontine syndrome (Millard-Gübler syndrome) /778
Inferior Foville syndrome or pontine Foville"/ 778 Bulbar syndromes / 778
Lateral bulbar, Wallenberg or artery syndrome posterior and inferior cerebellar / 778
Anterior bulbar syndrome / 779
Posterior bulbar syndromes / 779 Cerebellar syndrome / 779
Concept/ 779
General considerations / 779
Anatomophysiological count /779
Archicerebellum / 779
Paleocerebellum / 780
Neocerebellum / 781
Syndromography or positive diagnosis / 781 Clinical picture / 781
Topography of cerebellar syndrome / 783
Cerebellar or static vermis syndrome / 783
Hemispheres or kinetic syndrome / 783 Mixed syndrome / 783
Etiology / 783
Meningeal syndrome/783
Concept / 783
Brief anatomical account /783
Syndromography or positive diagnosis / 784
Clinical picture / 784
Complementary exams / 784
Etiology / 784
Intracranial hypertension syndrome / 785
Concept / 785
Syndromogenesis or pathophysiology / 785
Syndromography or positive diagnosis / 785
Clinical picture / 785
Complementary exams / 787
Etiology / 787
Comatose syndrome / 788
Concept/ 788
Syndromogenesis or pathophysiology / 788
Syndromography or positive diagnosis / 788
Examination of a patient in a coma / 790 Glascow Coma Scale / 790

- Etiology /791

55 Digestive system. Anatomy, physiology, general pathophysiology and clinical history / 792

- Anatomy and physiology / 792
  - Neoanatomical classification / 792
    - Upper or ingestion segment / 792
    - Middle or digestion segment / 792
    - Lower or excretion segment / 793
  - Gastric anatomy and histology / 794
    - Mucosa / 794
    - Muscularis mucosae / 796
    - Submucosa / 796
    - Muscular / 796
    - Serous / 796
  - Gastric physiology / 796
    - Motor skills / 797
      - Reservoir function / 797
      - Mixing function / 797
      - Empty function / 797
      - Pathophysiological consequences / 798
    - Gastric secretion / 798
      - Gastric juice / 799
  - Anatomy and physiology of the gallbladder and bile ducts / 801
    - Anatomy / 801
    - Physiology/801
  - Pancreatic function / 802
- General pathophysiology / 803
  - Motor disorders / 803
  - Secretory and absorption alterations / 803
  - Sensory alterations / 804
  - Mixed alterations / 804
  - Alterations in chewing, swallowing and salivary secretion / 804
- Medical history /804
  - Anamnesis / 805
    - Personal identity data / 805
      - Origin and nationality / 805
      - Age / 805
      - Sex/ 805
      - Profession / 806
    - Toxic habits / 806
  - Physical examination / 806
  - Additional investigations / 806

56 Digestive system. Symptoms of the oropharyngoesophageal segment / 807

- Sialorrhea/ 808
  - Concept / 808
  - Semiogenesis or pathophysiology / 808
    - Salivary reflex excitability /808
    - Irritability of the salivary glands themselves / 809
  - Semiodiagnosis / 809
- Xerostomia / 811
  - Concept / 811
  - Semiogenesis or pathophysiology / 811
  - Semiotechnics / 811
  - Semiodiagnosis / 811
- Lingual or buccolingual burning / 812
  - Concept / 812
  - Semiogenesis or pathophysiology / 812
  - Semiodiagnosis / 812
- Halitosis /812
  - Concept / 812
  - Semiogenesis or pathophysiology / 812
  - Semiotechnics /813
  - Semiodiagnosis / 813
- Taste alterations / 813
  - Concept / 813
  - Semiogenesis or pathophysiology / 813
  - Semiodiagnosis / 814
- Bruxism/815
- Dysphagia / 815
  - Anatomical summary of the esophagus / 815
  - Histological summary of the esophagus / 816
  - Physiological summary of swallowing / 816
  - Concept / 816
  - Semiogenesis or pathophysiology / 818
    - Pathophysiology of organic dysphagia / 818
    - Pathophysiology of functional dysphagia / 818
      - High functional dysphagia / 818
      - Medium functional dysphagia / 818
      - Low functional dysphagia / 818
  - Semiography /819
    - Location /819
    - Periodicity / 819
    - Intensity / 819
    - Quality /819
    - Start mode / 820
    - Associated symptoms / 820
    - Course / 820
  - Semidiagnosis / 820
- Heartburn / 822
  - Concept / 822
  - Semiogenesis or pathophysiology / 822
    - Intimate mechanism /822
  - Semiodiagnosis / 822
- Pituita / 823
  - Concept / 823
  - Semiogenesis or pathophysiology / 823
  - Semiodiagnosis / 823

57 Digestive system. Symptoms of the gastroduodenhepatobiliopancreatic segment /

- Major symptoms or capital symptoms / 825
  - Abdominal pain / 825
    - Preliminary considerations / 825
    - Concept / 826
    - Semiogenesis or pathophysiology / 826
      - Theory of the viscerosensitive reflex, by Ross and Mackenzie / 827
      - Theory of pure visceral pain /827

Theory of the peritoneum-sensitive reflex, by Morley / 829
Theory of vagal transmission of the reflex / 829
Intimate mechanism or visceral initiation of the pain reflex / 829
Eclectic pathogenic theory / 829
Semiography and semidiagnosis / 830
Vomiting / 845
Concept /845
Semiogenesis or pathophysiology / 845
Peripheral vomiting /845
Central vomiting /846
Intimate mechanism of gastric emptying in the vomit / 846
Semiography and semidiagnosis / 846
Clinical characteristics of peripheral and vomiting central /846
Hematemesis /851
Concept / 851
Differential diagnosis / 851
Semiogenesis or pathophysiology /852
Semiography and semidiagnosis / 852
Small symptoms or dyskinetic symptoms / 853
Hypersthenic dyskinetic symptoms / 853
Aerophagia and aerophagic belching / 853
Concept / 853
Semiogenesis or pathophysiology / 853
Semiography /853
Semiodiagnosis / 853
Gastric regurgitation / 855
Concept / 855
Semiogenesis or pathophysiology / 855
Semiography /855
Semidiagnosis / 855
Non-aerophagic belching / 855
Concept / 855
Semiogenesis or pathophysiology / 855
Semidiagnosis / 855
Gastric burning / 856
Concept / 856
Semiogenesis or pathophysiology / 856
Semiography and semidiagnosis / 856
Hypo /856
Concept / 856
Semiogenesis or pathophysiology / 856
Semiography and semidiagnosis / 857
Dyskinetic hyposthenic symptoms / 857
Gastric fullness, heaviness and satiety / 857
Concept / 857
Semiogenesis or pathophysiology / 857
Semiography and semidiagnosis /857
Nausea / 857
Concept / 857
Semiogenesis or pathophysiology / 858
Semiography and semidiagnosis / 858
Epigastric jump / 858
Concept /858
Semiogenesis or pathophysiology / 858
Semiography and semidiagnosis / 858 58
đigestívo system. Symptoms of the enterocollicorrectal segment / 859
Major symptoms or capital symptoms / 860
Enterorrhagia and melena /860
Concept /860
Semiogenesis or pathophysiology/ 860
Semidiagnosis /861
Semiological significance of enterorrhagia /861
Semiological significance of the mane/861
Constipation /861
Concept /861
Semiogenesis or physiopathology /861
Decreased permeability of the digestive tract (in particular of the small and large intestine) / 862
Neuromotor balance alterations / 862
Alterations of humoral agents of peristatic regulation / 863
Decrease in water intake /863
Hyperactivity of the digestive enzymatic action and intestinal hyperabsorption /863
Lack of waste substances in the diet / 863 Accessory factors / 863
Semiography/863
Semiodiagnosis /864
Diarrhea/ 865
Concept / 865
Semiogenesis or pathophysiology / 865
Alteration of the intestinal mucosa / 865 Alteration of neuromotor balance / 867 Decrease in enzymatic action / 867 Decrease in intestinal absorption / 867 Dietary alterations / 868
Semiography / 868
Semiodiagnosis / 869
Small symptoms or dyskinetic symptoms 870
Abdominal distension or bloating / 870
Concept / 870
Semiogenesis or pathophysiology / 870
Semiodiagnosis / 870
Borborygmos /872
Concept / 872
Semiogenesis or pathophysiology / 872
Semiodiagnosis / 872
Expulsion of gases through the anus / 872
Colic heartburn / 872
Particular symptoms of the rectum and anus / 872
Rectal pain or proctalgia / 873
Concept / 873
Semiogenesis or pathophysiology / 873
Semiography /873
Semiodiagnosis / 873
Rectal bleeding / 873
Concept /873
Semiogenesis or pathophysiology / 873
Semiodiagnosis / 874
Rectal flow / 874

Concept / 874
Semiogenesis and semidiagnosis / 874
Anal itching / 874
Concept /874
Semiogenesis or pathophysiology / 874
Semiodiagnosis / 874
Rectal heaviness and protrusion / 874
Concept, semiogenesis and semiography / 874 Anal
burning / 874 Semiogenesis or pathophysiology / 861
Decreased permeability of the digestive tract (particularly the small and large intestine) / 862 Alterations in neuromotor balance / 862 Alterations in the humoral agents of peristaltic regulation / 863

Decrease in water intake / 863
Hyperactivity of digestive enzyme action and intestinal hyperabsorption / 863
Lack of waste substances in the diet / 863 Accessory factors / 863
Semiography /863
Semiodiagnosis / 864
Diarrhea/ 865
Concept, semiogenesis and semiography / 874

59 Digestive semiology in general and regional physical examinations /875

Digestive semiology in the general physical examination /875 Attitude
and recumbency / 875
Constitution / 876
Weight and height / 876
Facies / 876
Skin color / 877
Extremities / 877
Digestive semiology in the regional physical examination/ 878
Physical examination of the mouth / 878
Introduction / 878
Semiogenesis or pathophysiology / 878
Semiography and semidiagnosis / 878
Closed mouth (external examination): lips and corners / 878
Jugal mucosa / 879
Language / 880
Teeth / 887
Gums / 888
Hard and soft palate / 888
Salivary glands / 888
Physical examination of the oropharynx /888
Physical examination of the esophagus /888

60 Alterations in the regional physical examination of the abdomen /889

Pathological data obtained by isolated physical methods / 889
Symptoms identifiable on inspection / 889
Skin disorders / 890
Alterations of the contour or pathological typology / 890
Alterations of the navel / 890
Volume changes / 890
Global increases in the abdomen / 891 Partial
increases in the abdomen / 892
Abnormal movements /892
Symptoms identifiable on palpation / 892
Pathological data collected during palpation of the abdominal wall / 892
Pathological data collected during palpation of the abdominal cavity (abdominal tension and viscera) / 893

Symptoms identifiable on percussion / 895
Hypertympanism / 895
Hypotympanism / 895
Dullness /895
Symptoms identifiable on auscultation / 896 Combined
physical methods in abdominal semitechnics / 896 Exploration of a free
ascites / 896 Collateral venous circulation / 896

61 Particular physical examination of the abdomen by areas and organs /897

Examination of the stomach and epigastrium / 897
Clinical anatomy / 897
Semiotechnics / 898
Normal data / 898
Pathological data / 898
Examination of the liver, gallbladder, pancreas and right upper quadrant / 899
Clinical anatomy / 899
Liver / 899
Gallbladder /900
Pancreas / 900
Semiotechnics / 900
Palpation of the liver / 900
Simple or single-handed palpation / 900
Chauffard or rally bimanual palpation / 900

Gilbert's bimanual palpation / 900
Mathieu bimanual palpation / 901
Devotee's Palpation / 901
Glenard bimanual palpation (thumb palpation) / 901
Palpation of the gallbladder / 902
Murphy maneuver / 902 Abraham
maneuver / 902 Fiessinger
maneuver /902 Palpation of the
pancreas / 902 Grott method / 903

Mallet-Guy method /903
Desjardins pancreatic point / 903 Liver
percussion / 904
Normal data / 904
Pathological data / 904
Spasticity or parietal contracture / 904
Hepatoptosis or descent of the liver / 905
Hepatomegaly / 905
Caused pain /909
Vesiculomegaly or large palpable vesicles / 909
Increased volume of the pancreas / 911
Vascular beats / 911
Examination of the transverse colon, jejunum, ileum and mesogastrium / 911

Clinical anatomy /911
Normal data / 912
Pathological data / 912
Examination of the ascending colon and the void or right flank /913
Clinical anatomy /913
Normal data / 913
Pathological data /913
Examination of the descending colon and the void or left flank /913
Clinical anatomy /913
Normal data / 913
Pathological data /913
Examination of the sigmoid loop of the colon and hypogastrium /913
Clinical anatomy /913
Normal data / 913
Pathological data /913
Examination of the lower portion of the descending colon, the sigmoid loop and the left iliac fossa /913
Clinical anatomy / 913
Normal data /914
Pathological data /914
Examination of the cecum, appendix and right iliac fossa /914
Clinical anatomy / 914
Normal data /914
Pathological data / 914
Examination of the rectum and anus /918
Pathological data / 918
Inspection / 918
Rectal touch /918

62 Digestive system. Additional investigations /919

Paracentesis (ascitic puncture) / 919
Definition /919
Indications and contraindications / 920
Material/920
Technical procedure / 920
Possible incidents and accidents / 921
Additional imaging exploration / 921
Simple x-ray /921
Contrast radiography / 921
Ultrasonography / 921
Computed axial tomography (CAT) /922
Nuclear magnetic resonance / 922
Radioisotope imaging / 922
Endoscopic methods / 922
Laparoscopy or peritoneoscopy / 923 Definition / 923
History /923
Applied anatomy and physiology / 923
Anatomy / 923
Physiology /923
Indications / 923
For diagnostic purposes / 923 For therapeutic purposes / 925
Contraindications / 925
Normal data / 925
Pathological data / 926
Peritoneum / 926
Liver / 926
Gallbladder /928
Stomach /928
Jejunum and ileum / 929
Large intestine /929
Spleen /929
Retroperitoneal organs and formations / 929
Complementary examinations that can be performed through laparoscopy / 929
Directed biopsy /929
Laparoscopic cholangiography /930
Splenoportography with manometry /930
Cystography (cystography) /930
Laparophotography / 930
Endoscopic surgery / 930
Upper gastrointestinal endoscopy / 930
Esophagoscopy / 930
Concept and importance / 930
Indications for diagnostic purposes / 930
Indications for therapeutic purposes / 932
Contraindications / 932
Equipment (esophagoscopes) / 932
Technical procedure / 932
Normal data /932
Pathological data / 933
Complications /935
Gastroscopy / 935
Indications for diagnostic purposes / 935
Indications for therapeutic purposes / 935
Contraindications / 935
Teams / 935
Technical procedure / 937
Endoscopic guidance / 937
Normal data /937
Pathological data /937
Duodenoscopy / 942
Team / 942
Indications for diagnostic purposes / 942
Indications for therapeutic purposes / 942
Panendoscopy / 943
Endoscopic cholangiopancreatography (EPC) / 943
Indications for diagnostic purposes /943
Indications for therapeutic purposes / 944
Endoscopies of the rectum and colon /944
Rectosigmoidoscopy / 944
Concept / 944
Indications and contraindications / 944
Equipment /
Semiotechnics / 945
Normal data / 946
Pathological data /946
Colonoscopy / 948
Concept /948
Indications for diagnostic purposes /950 Indications for therapeutic purposes / 950 Contraindications / 950
Teams /952

Semiotechnics / 952
Exploratory methods by probing / 952
Measurement of gastric acidity (gastrochymogram) / 952
Indications / 952
Contraindications / 952
Normal data / 953
Pathological data / 953
Kay test /954
Hollander test /954
Biliary drainage or Meltzer-Lyon/954 test
Indications /954
Contraindications / 954
Normal data / 954
Pathological data /956
Timed Biliary Drainage / 957
Elimination of bromosulfthalein (BSF) in bile /957 Exploration of pancreatic function / 957
Normal data / 957
Other studies using catheters or probes / 958 Esophageal manometry / 958
Pathological data /958
esophageal pH-metry / 958
Blind gastric biopsy / 958
Peroral biopsy of the small intestine / 958 Colonic manometry / 958

63 Main syndromes of the digestive system / 960

Gastrointestinal hormones / 960
Gastrin /961
Secretin / 961
Cholecystokinin / 961
Insulin/961
Glucagon /961
Gastric inhibitory polypeptide /962
Motilin / 962
Pancreatic polypeptide / 962
Vasoactive intestinal polypeptide / 962
Enteroglucagon / 962
Jaundice syndrome / 962
Definition and concept / 962
Syndromography or positive diagnosis / 963
Clinical picture / 963
Complementary exams / 963
Differential diagnosis /963
Syndromogenesis or pathophysiology (pathogenic diagnosis) / 964
Jaundice due to increased production of free or unconjugated bilirubin that reaches the liver cell / 964
Clinical picture / 964
Complementary exams /964
Jaundice due to disorders in the uptake of free bilirubin by the hepatocyte /964
Clinical picture and complementary examinations / 964
Jaundice due to disorders in the conjugation of bilirubin within the hepatocyte /964
Clinical picture / 966
Complementary exams /966
Jaundice due to disorders in the excretion of conjugated bilirubin, from the hepatocyte to the duodenum /966
Intrahepatic cholestasis / 966
Extrahepatic cholestasis / 968
Etiology (etiological diagnosis) /969 Ascitic syndrome/969
Concept / 969
Syndromography or positive diagnosis / 970
Clinical picture /970
Functional symptoms / 970
Physical signs / 970
Other symptoms / 971
Complementary examinations /971
Radiological examinations /971
Abdominal ultrasound / 971
Laboratory examinations /971
Laparoscopy /971
Differential diagnosis / 973
Abdominal conditions / 973
Pelvic conditions /973
Syndromogenesis or pathophysiology / 973
Etiology /976
Chronic diarrheal syndromes / 976 Malabsorption syndrome / 976
Concept / 976
Rating /978
Primary malabsorption syndromes / 978 Secondary or symptomatic malabsorption syndromes / 978
Syndromogenesis or pathophysiology / 978
Deficiency of cofactors necessary for the preparation of nutrients to be absorbed / 978 Shortening of the contact time of nutrients with the mucosa / 978
Decrease in the intestinal absorption surface /978
Decrease in the digestive enzymes of the intestinal mucosa / 978
Disorders of specific transport mechanisms / 978
Disorders of vascular outflow from the intestine / 979
Presence of abnormal flora /979
Syndromography or positive diagnosis /979
Clinical picture /979
Complementary exams /979
Etiology / 980
Dysenteric syndrome / 980
Concept / 980
Syndromogenesis or pathophysiology / 980
General mechanisms /980
Specific mechanisms / 981
Syndromography or positive diagnosis /981 Clinical picture /981
Complementary exams / 982
Etiology / 982
Digestive bleeding / 982

Concept / 982
Syndromogenesis or pathophysiology / 983
Syndromography or positive diagnosis /983 Clinical picture /983
Complementary exams / 983 Etiology /985
64 Hemolymphopoietic system. Hematopoiesis and main symptoms and signs /986
Hematopoiesis / 986
Organization of hematopoietic cells /986 Compartmental model of hematopoiesis / 986 Erythropoiesis /988
Leukopoiesis. /989
Granulocytic series: neutrophils, eosinophils and basophils / 989
Agranulocytic series: monocytic and lymphocytic /989
Monocytic series / 989
Lymphocytic series /990
Megakaryopoiesis / 992
Plasma cells /992 Main symptoms and signs / 993
Cutaneomucosal pallor / 993
Glossitis /993
Bone Pain / 993
Purple / 993
Bruises / 993
Increased volume of the spleen and lymph nodes / 993
Neurological manifestations / 994
65 Main syndromes of the system hemolymphopoietic /995
Anemic syndrome /995
Concept /995
Normal physiology of erythropoiesis /995
Pathophysiological classification /995
Disorders in the multipotential stem cell and unipotential / 996
Disorders in DNA synthesis /996
Disorders in heme synthesis / 996
Disorders in globin synthesis / 996 Disorders in enzymatic synthesis of red blood cells /997
Disorders due to external interference in bone marrow functions / 997
Disorders due to interference with the survival of red blood cells / 997
Syndromography or positive diagnosis /997
Clinical picture /997
Complementary exams / 998
Adenic syndrome /998
Concept /998
Syndromogenesis or pathophysiology / 998
Syndromography or positive diagnosis /998
Differential diagnosis / 999
Etiology /999
Splenomegalic syndrome / 1000
Concept / 1000
Syndromography or positive diagnosis / 1001
Clinical picture / 1001
Data obtained from the anamnesis / 1001 Data from the physical examination of the spleen / 1001
Differential diagnosis / 1002
Clinical interpretation of splenomegaly / 1002
Pathophysiology and etiology / 1003
Purpuricohemorrhagic syndromes / 1003
Concept /1003
General physiology of hemostasis / 1003
Normal coagulation mechanism / 1004
Pathophysiological classification / 1006
Hemorrhagic diatheses due to disorders in the plasma coagulation mechanisms and excessive circulating anticoagulants / 1006 Hemorrhagic diatheses due to platelet disorders / 1006 Hemorrhagic diatheses due to vascular disorders /1006 Hemorrhagic diatheses of complex etiology / 1006 Syndromography or positive diagnosis / 1006 Etiology / 1008
66 Urinary system. Anatomy, physiology, semiology and complementary exams /1009
Anatomy and physiology / 1009
Semiology of the urinary system / 1012
Personal identity data / 1012
Age / 1012
Sex/ 1012
Occupation / 1012
Next anamnesis / 1012
Home/ 1012
Remote anamnesis / 1014
Previous diseases / 1014
Main symptoms and signs / 1014 Pain / 1014
Simple or capsular pain / 1014
Nephritic colic / 1015
Hematuria/ 1018
Concept/ 1018
Semiogenesis / 1018
Semiotechnics and semiography / 1018
Semiodiagnosis / 1018
Urine evacuation disorders / 1018
Polyuria / 1020
Oliguria and anuria / 1020
Nocturia/ 1023
Opsyuria / 1023
Frequencyuria / 1023
Dysuria / 1023
Bladder urgency / 1023
Overflow urination/ 1023
Urinary incontinence / 1023
Changes in urine color / 1023
Changes in urine density / 1024
Alterations to the physical examination / 1024
Inspection / 1024
Palpation / 1024

Increased kidney volume (nephromegaly) /1025 Renal ureteral pain points / 1025
Presence of edema / 1025
Percussion / 1025
Auscultation / 1025
Complementary examinations / 1025
Laboratory examinations /1025
Endoscopic examinations / 1027
Imaging examinations / 1027
Simple x-ray or simple urinary tract (TUS) / 1027
Abdominal ultrasound / 1027
Excretory urography or descending urogram / 1027
Ascending pyelography / 1027
Cystography and urethrocystography / 1027
Retropneumoperitoneum / 1028
Aortography / 1028
Cavography / 1028
Computed axial tomography (CAT) / 1028
Magnetic resonance imaging (MRI) / 1028
Scintigraphy/ 1028
Histological examinations through biopsies / 1028

67 Main syndromes of the urinary system / 1029
Acute renal failure syndrome / 1029 Concept / 1029
Syndromogenesis or pathophysiology / 1029
Syndromography or positive diagnosis /1029
Clinical picture / 1029
Background / 1029
Symptoms / 1030
Complementary exams / 1030
Etiology/ 1030
Chronic kidney failure syndrome 1030
Concept / 1030
Syndromogenesis or pathophysiology / 1030
Theory of intact nephrons / 1032
Theory of pathogenic mechanisms of uremic manifestations / 1032
Syndromography or positive diagnosis / 1032
Clinical picture / 1032
Background / 1032
Symptoms / 1032
Complementary exams / 1032
Etiology/ 1035
Nephrotic syndrome / 1035
Concept / 1035
Syndromogenesis or pathophysiology / 1035
Syndromography or positive diagnosis / 1038 Clinical-humoral picture / 1038
Proteinuria / 1038
Hypoproteinemia and other protein disorders plasma / 1038
Hyperlipidemia/1038
Edema / 1038
Etiology / 1038

68 Aqua and electrolytes. Preliminary clarifications and hydroelectrolyte balance /1042
Preliminary clarifications / 1042
Concepts / 1042
Weight / 1042
Mass / 1042
Atom / 1042
Atomic structure / 1042
Proton / 1043
Neutron / 1043
Electron / 1043
Atomic weight (p.a.) / 1043
Atom-gram / 1043
Molecule / 1043
Molecular weight (m.p.) / 1043
Molecule-gram (mol) / 1043
Chemical element / 1043
Isotope / 1043
Valencia / 1043
Units of measurement / 1043
Volume units / 1044
Chemical Combining Power Unit / 1044
Conversion from milligrams (mg) to milliequivalents (mEq)/ 1045
Electrolyte concentration unit / 1045 Molar solution / 1045
Conversion between milligrams per cent and millimoles per liter/1046
Osmotic pressure unit / 1046
Osmolality and osmolarity / 1047
Water balance / 1047
Regulation of water and salts / 1047
Gains or income from body fluids /1048 Losses or income from body fluids / 1048 Summary / 1049
Neurohormonal control of water / 1050
Electrolyte balance / 1052
Regulation of the main electrolytes / 1052
Chlorine and sodium / 1052
Potassium / 1055
Magnesium / 1055
Calcium / 1055
Phosphorus/ 1056

69 Alterations in water, electrolyte and acid-base balances /1057
Alterations in water balance / 1057
Default water imbalance: dehydration / 1057 Previous considerations / 1057
Hypertonic dehydration syndrome / 1058 Synonymy/ 1058
Concept / 1058
Syndromogenesis or pathophysiology / 1058
Syndromography or positive diagnosis / 1060
Etiology / 1060
Hypotonic dehydration syndrome / 1061 Synonymy/ 1061
Concept / 1061
Syndromogenesis or pathophysiology / 1061

Syndromography or positive diagnosis / 1061
Etiology / 1063
Mixed dehydration syndrome / 1063
Synonymy/ 1063
Concept/ 1063
Syndromogenesis or pathophysiology / 1063
Syndromography or positive diagnosis / 1064
Etiology / 1064
Water imbalance due to excess: hyperhidrotation / 1064
Hyperhydration syndrome / 1064
Synonymy/ 1064
Concept / 1064
Syndromogenesis or pathophysiology / 1064
Syndromography or positive diagnosis / 1064
Etiology / 1064
Electrolyte balance alterations: excess and defect / 1064
Chlorine imbalance / 1065
Hypochloremia syndrome / 1065
Syndromography or positive diagnosis/ 1065
Etiology/ 1065
Hyperchloremia syndrome / 1065
Syndromography or positive diagnosis / 1065
Etiology / 1065
Sodium imbalance / 1066
Hyponatremia syndrome / 1066
Syndromography or positive diagnosis / 1066
Pathophysiology and etiology / 1066
Hypernatremia syndrome / 1066
Syndromography or positive diagnosis / 1066
Classification / 1066
Syndromogenesis and etiology / 1066
Potassium imbalance / 1067
Hypokalemia syndrome / 1067
Syndromography or positive diagnosis / 1067
Etiology / 1068
Hyperkalemia syndrome / 1068
Syndromography or positive diagnosis / 1068
Etiology / 1068
Magnesium imbalance/ 1068
Hypomagnesemia syndrome / 1068
Syndromography or positive diagnosis / 1068
Etiology / 1068
Hypermagnesemia syndrome / 1068
Syndromography or positive diagnosis / 1068
Etiology / 1068
Calcium and phosphorus imbalance / 1068
Hypocalcemia syndrome / 1069
Syndromography or positive diagnosis / 1069
Etiology / 1069
Hypercalcemia syndrome / 1069
Positive diagnosis, etiology and clinical picture / 1069
Alterations in acid-base balance /1069
Regulation of acid-base balance / 1069 Definitions of acidosis and alkalosis / 1070
Laboratory studies in acid-base imbalance /1071 Hemogasometry / 1071
pH/ 1071
Partial pressure of CO, (pCO,) / 1071 Base buffer (BB) / 1071
Standard bicarbanate (BS) / 1071
Base excess (EB) / 1071
Fundamental parameters for the study of acid-base balance / 1072
Hydrogen ion concentration [H+] / 1072
PCO, blood and bicarbonate calculation / 1073 Remaining anions (RA) / 1073
Classification of alterations in acid-base balance / 1074
Expected compensation ranges in simple disorders / 1074
Acidosis syndrome / 1074 Metabolic acidosis / 1074 Respiratory acidosis / 1077 Alkalosis syndrome / 1078 Metabolic alkalosis / 1078
Respiratory alkalosis / 1079

70 Semiology of the male and female reproductive systems /1081
Male reproductive system / 1081
Interrogation Anamnesis 1081
Most frequent pathological processes / 1081
Alterations detectable on physical examination / 1084
Inspection / 1084
Palpation / 1084
Palpation of the scrotum / 1084
Palpation of the tunica serosa vaginalis / 1084
Palpation of the testicle and epididymis / 1084
Palpation of the vas deferens and spermatic cord / 1084
Enlargement of the prostate / 1089 Palpation of the urethra / 1089
Female reproductive system / 1089
Interrogation or Anamnesis / 1089
Most frequent gynecological symptoms / 1089 Pain / 1089
Semiogenesis or pathophysiology / 1089
Semiotechnics / 1089
Semiography / 1092
Semiodiagnosis / 1093
Menstrual disorders / 1093
Concept/ 1093
Semiogenesis or pathophysiology / 1093
Semiotechnics / 1094
Semiography/ 1094
Semiodiagnosis / 1094
Amenorrhea / 1094
Concept / 1094
Semiogenesis and semidiagnosis / 1095
Semiotechnics / 1095
Semiography/ 1095
Leucorrhoea / 1096
Concept / 1096
Semiogenesis / 1096
Semiotechnics / 1096

Semiography and semidiagnosis / 1096
Alterations in the physical examination / 1098
Semiology of the breasts / 1098 Increase
in volume of the uterus / 1098
Concept / 1098
Semiogenesis or pathophysiology / 1099
Semiotechnics /1099
Semiography/ 1099
Semiodiagnosis / 1099
Increased volume of the tubes / 1100 Concept
/ 1102
Semiogenesis and semidiagnosis / 1102
Semiotechnics /1102
Semiography /1102
Increase in ovary volume / 1 102
Concept/ 1102
Semiogenesis and semidiagnosis / 1105
Semiotechnics / 1105
Semiography / 1105

71 Tabolic endocrinome system.
/ 1109 Neuroendocrine regulation and main symptoms

Neuroendocrine regulation / 1109
Nature of a hormone / 1110
Retrocontrol or feedback of secretion
hormonal / 1110
Chemistry of hormones / 1110
Intracellular hormonal mediators / 1111
Mechanisms involved in the different categories of
endocrine diseases / 1111
Primary hyperfunction of the endocrine glands / 1111 Secondary
hyperfunction of the endocrine glands / 1112 Insufficiency of the
endocrine glands / 11 12 "Functional" disorders of the
endocrine glands / 1112 Insufficiency of response of an end organ
to some hormone / 1112

Production of an abnormal hormone by an endocrine gland / 1112

Production of hormones by a non-endocrine organ / 1112
Iatrogenic endocrine disease / 1113
Role of the hypothalamus in endocrine regulation / 1113 Control of
biogenic amines on hypophysiotropic functions / 1114

Main symptoms of the endocrinometabolic system / 1114
Asthenia / 1114
Concept / 1114
Semiography / 1114
Semiodiagnosis / 1115
Polyuria /1115
Polydipsia /11 15
Semiogenesis and semidiagnosis / 1115
Polyphagy /1 116
Semiodiagnosis / 1116
Obesity / 1116
Synonymy/ 11 16
Concept / 1116
Semiogenesis or pathophysiology / 1116
Endogenous factors / 1116
Exogenous psychological factors / 1118
Semiography / 1119
Ideal weight / 1119
Clinical study / 1119
Semiodiagnosis / 1120
Thinness / 1 120
Concept / 1120
Semiography/1120
Semiogenesis and semidiagnosis / 1120
Thinness due to incorrect diet / 1120 Thinness due to
illness / 1121
Thinness by constitutional state / 1121 Increased
thyroid volume (Goiter) / 1121 Concept / 1121

Semiogenesis or pathophysiology / 1121
Functional hypertrophy and hyperplasia / 1122
Inflammatory reactions / 1l122
Neoplasms / 1122
Semiotechnics / 1122
Marañón Maneuver / 1122
Measurements / 1122
Semiography and semidiagnosis / 1122
Simple colloid goiter / 1122
Nodular goiter or adenomatous goiter / 1122
Iatrogenic goiter / 1122
Family goiter / 11 24
Graves-Basedow Disease / 1124
Acute infectious thyroiditis / 1124
Chronic thyroiditis/ 1 124
Neoplasms / 1124
Exophthalmia or exophthalmos/1124
Concept / 1124
Semiogenesis, semiography and semidiagnosis / 1124
Bilateral exophthalmia / 1124
Unilateral exophthalmia /1124
Height alterations / 1129 Short stature
syndrome / 1130
Concept / 1130
Syndromogenesis or pathophysiology / 1130
Etiological classification / 1130
Pituitary short stature syndrome / 1 131 True
dwarfism or nanosomia / 1131 Hypothyroid
dwarfism: congenital myxedema and sporadic cretinism /
1131
Endemic cretinism / 1131
Achondroplasia or chondrodystrophy / 1131
Short pituitary stature with hypogonadism / 1133
Short stature with Turnerian gonadal dysgenesis (
gonadal dysgenesis or Turner syndrome) / I133 Tall
stature syndrome / 1133
Etiological classification / 1133
Pituitary gigantism / 1133
Precocious sexual development and adrenogenital syndrome /
1133 Tall stature with hypogonadism / 1133
Congenital generalized lipodystrophy / 1133
Cerebral gigantism or Sotos syndrome / 1133

Hirsutism / 1134
Concept /1134
Hair categories / 1134
Semiogenesis or pathophysiology / 1134
Semiography/ 1134
Clinical picture / 1134
Semiodiagnosis / 1134
72 Endocrinemetabolic system. pituitary and pituitary syndromes /1137
Anatomy and histology of the pituitary / 1137
Portal circulation of the pituitary / 1138
Result of hypophysectomy / 1139
Histology of the pituitary / 1139
Physiology of the pituitary / 1141
Anterior pituitary (adenohypophysis) / 1141
Anteropituitary hormones / 1141
Growth hormone or somatotropin (GH) / 1142 Thyrotropin, thyrotropic hormone or thyroid stimulating hormone (TSH) /1142
Adrenocorticotropin or corticotropin (ACTH) / 1143 Follicle-stimulating hormone (FSH) / 1143 Luteinizing hormone or interstitial cell-stimulating hormone (LH) / 1 143

Prolactinau lactogenic hormone (PRL former LTH) / 1144
Melanocyte-stimulating peptides (former MSH) / 1144

Control of anterior pituitary secretion / 1144 Hypothalamic neurosecretion: hypophysiotropic neurons / 1145
General considerations of pituitary syndromes / 1145 Lesions affecting the pituitary gland / 1147
Anterior lobe syndromes / 1148
Hyperpituitarism syndromes / 1148
Concept/ 1148
Rating / 1148
Injury or organic hyperstimulation syndromes /
Reactive or hyperstimulation syndromes functional / 1148
Symptoms and complementary tests / 1149
Adrenal gland / 1149
Thyroid gland / 1 149
Sexual glands (ovaries and testicles) / 1149
Acromegaly / 1149
Concept / 1149
Syndromogenesis or pathophysiology / 1149
Syndromography or positive diagnosis / 1150
Etiology / 1152
Pituitary gigantism / 1152
Concept / 1152
Syndromogenesis or pathophysiology / 1152
Syndromography or positive diagnosis / 1152
Etiology / 1153
Hypopituitarism syndrome: pituitary insufficiency /1153
Concept / 1153
Syndromogenesis or pathophysiology / 1153
Syndromography or positive diagnosis / 1153 Clinical picture / 1153
Complementary exams / 1154
Etiology / 1154
Posterior lobe syndrome / 1159 Diabetes insipidus / 1159 Concept / 1159
Syndromogenesis or pathophysiology / 1159
Physiological summary / 1159
Mechanism of polyuria insipidus / 1160 Mechanism of symptoms / 1160
Syndromography or positive diagnosis / 1160
Clinical picture / 1160
Complementary exams / 1161
Etiology / 1164

73 Endocrinemetabolic system. Thyroid and thyroid syndromes /1165
Thyroid anatomy / 1165
Thyroid embryology / 1165
Thyroid histology / 1 166
Thyroid physiology / 1166
Thyroid hormones / 1166
Iodine metabolism / 1168
Thyroid regulatory system / 1170
Effector organs /1170
Thyroid hormone blocking agents / 1170 Functions of thyroid hormones / 1170
Tissue oxidation function / 1170
Metabolic functions / 1170
Function on the circulatory system / 1170 Function on the digestive system / 1171 Function on the renal system / 1171 Function on the nervous system / 1171 Function on the muscular system / 1171 Function on the integumentary tissue / 1171
Other functions / 1171

Thyroid syndromes / 1171
Hyperthyroid syndrome (hyperthyroidism) / 1172
Concept / 1172
Synonymy / 1 172
Syndromogenesis or pathophysiology /1172
Syndromography or positive diagnosis / 1172 Clinical picture /1178
Complementary examinations / 1178
Etiology /1 178
Hypothyroid syndrome / 1178
Concept/ 1178
Syndromogenesis or pathophysiology / 1178
Syndromography or positive diagnosis / 1178
Clinical picture / 1178
Respiratory system/ 1182
Complementary exams / 1183
Etiology /1183
Juvenile hypothyroidism / 1183
Congenital hypothyroidism (thyreosis) /1185

74 Tabolic endocrinome system. Parathyro ides and parathyroid syndromes / 1186
Parathyroid anatomy /1186

- Parathyroid histology / 1186
- Physiology of the parathyroids / 1186
  - Calcium / 1187
  - Phosphorus / 1187
  - Parathyroid hormone / 1189
  - Calcitonin / 1189
  - Vitamin D, or cholecalciferol/ 1189
  - Magnesium /1189
- Parathyroid syndromes / 1190
  - Primary hyperparathyroid syndrome / 1190
    - Concept / 1190
    - Syndromogenesis or pathophysiology / 1190
    - Syndromography or positive diagnosis / 1192
      - Clinical picture / 1192
      - Complementary exams / 1194
    - Etiology / 1 194
  - Hypoparathyroid syndrome / 1194
    - Concept / 1194
    - Syndromogenesis or pathophysiology / 1194
    - Syndromography or positive diagnosis / 1196
      - Clinical picture / 1196
      - Complementary exams / 1200
    - Etiology / 1200

75 Endocrinemetabolic system. Adrenal glands and their syndromes /1201

- Anatomy of the adrenal glands / 1201
- Adrenal histology / 1201
- Physiology of the adrenals / 1204 Adrenal cortex / 1204
    - Function of mineralocorticoids / 1204
    - Function of glucocorticoids / 1204
    - Sex hormones / 1206
  - Adrenal medulla / 1206
  - Regulation of adrenal steroid secretion /1207
  - Steroid biosynthesis / 1207
- Adrenal syndromes / 1207
  - Pure metabolic hypercorticism: Cushing syndrome / 1208
    - Concept / 208
    - Syndromogenesis or pathophysiology / 1209
    - Syndromography or positive diagnosis / 1209
      - Clinical picture /1209
      - Complementary exams / 1211
    - Etiology / 1211
  - Pure androgenic hypercorticism: adrenogenital or Cooke-Apert-Gallais syndrome / 1211
    - Concept / 1211
    - Syndromogenesis and syndromography / 1211 21-hydroxylase deficiency /1211
      - 11-B-hydroxylase deficiency / 1213
      - Adrenal tumors / 1213
      - Late or postpubertal forms / 1213 Etiology of adrenogenital syndrome / 1213
  - Estrogenic hypercorticism: syndrome due to excess estrogen (feminization) / 1213
    - Syndromography or positive diagnosis / 1215
      - Clinical picture / 1215
      - Complementary exams / 1215
  - Hypermineralocorticism: primary hyperaldosteronism or Conn syndrome / 1215
    - Concept / 1215
    - Syndromogenesis or pathophysiology / 1215
    - Syndromography or positive diagnosis /121S
      - Clinical picture / 1215
      - Complementary exams / 1215
    - Etiology/ 1218
  - Hypermineralocorticism: secondary hyperaldosteronism / 1218
  - Acute adrenal corticosteroid insufficiency: Waterhouse-Friderischsen syndrome / 1218
    - Concept / 1218
    - Syndromogenesis or pathophysiology / 1218
    - Syndromography or positive diagnosis / 1218
      - Clinical picture / 1218
      - Complementary exams / 1218
    - Etiology /1218
  - Chronic adrenocortical insufficiency: Addison's disease / 1221
    - Concept / 1221
    - Syndromogenesis or pathophysiology / 1221
    - Syndromography or positive diagnosis /1221
      - Clinical picture / 1221
      - Complementary exams / 1224
    - Etiology/ 1224
  - Spinal cord hyperfunction syndrome (pheochromocytoma) / 1224
    - Concept / 1224
    - Syndromogenesis or pathophysiology / 1224
    - Syndromography or positive diagnosis / 1224
      - Clinical picture / 1227
      - Complementary exams/ 1227
    - Etiology/ 1227
  - Tumor syndrome / 1227
    - Syndromography or positive diagnosis / 1227
      - Clinical picture / 1227
      - Complementary exams / 1227

76 Endocrinemetabolic system. Syndromes hyperglycemic and hypoglycemic Yes /1228

- Hyperglycemic syndrome / 1229
  - Concept / 1229
  - Syndromogenesis or pathophysiology and positive diagnosis / 1229
    - Complementary exams / 1230 Etiology/ 1230
- Hypoglycemic syndrome / 1231
  - Concept / 1231
  - Syndromogenesis or pathophysiology / 1231
    - Hypoglycemia in the postprandial period / 1231
    - Fasting hypoglycemia during the metabolism of endogenous energy substances / 1232
      - Absolute spontaneous hypoglycemia / 1232
      - Relative spontaneous hypoglycemia / 1233
    - Induced hypoglycemia on an empty stomach / 1233 Syndromography or positive diagnosis / 1233
    - Clinical picture / 1233
      - Hypoglycemic crisis / 1235

Hypoglycemic coma / 1235
Complementary exams / 1236
Etiology / 1236

77 Endocrinemetabolic system. Gonadal syndromes /1237

Male hypogonadism syndromes / 1237
Primary testicular insufficiency / 1237
Secondary testicular insufficiency / 1238
Hypogonadism of unknown etiology with endocrine alterations / 1238
Eunuchoidism / 1238
Prepubertal eunocoidisms / 1238
Late or postpubertal geldings / 1238
Klinefelter syndrome / 1240
Syndromography or positive diagnosis / 1240
Pathophysiology and etiology / 1240
Gynecomastia / 1240
Syndromogenesis or pathophysiology / 1240
Male climacterium or andropause / 1240
Syndromography or positive diagnosis / 1240 Infertility and coeundi impotence / 1241 Female hypogonadism syndromes / 1243 Castration / 1243

Syndromography or positive diagnosis / 1243 Clinical picture / 1243
Complementary exams /1243
Pathophysiology and etiology / 1243
Menopause / 1243
Syndromography or positive diagnosis / 1243 Clinical picture / 1243
Complementary exams / 1243
Turner syndrome / 1243
Pathophysiology and etiology / 1243
Syndromography or positive diagnosis / 1245
Clinical picture / 1245
Complementary exams /1245
Noonan syndrome / 1245
Syndromogenesis or pathophysiology / 1245
Polycystic ovary syndrome (Stein-Leventhal) / 1245
Syndromogenesis or pathophysiology / 1245
Syndromography or positive diagnosis / 1245 Clinical picture / 1245
Complementary examinations / 1246
Intersexuality and hermaphroditism / 1246
Syndromography or positive diagnosis / 1246
Clinical picture / 1246
Complementary examinations / 1246
Hypergonadism syndromes / 1246
Male hypergonadism. Male hypergenitalism / 1246
Female hypergonadism / 1246
Hyperfolliculinism or hyperstrinism / 1246
Complementary exams /1248
Pathophysiology and etiology / 1248
Hyperluteism / 1248
Complementary exams /1248

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ALPHABETICAL INDEX OF SUBJECT

TO

Abdomen, normal, 44
  anatomy, 44
    ZONES, 44
      Anglo-Saxon school, 46
      french school, 45
      visceral projections by zones, 46
  auscultation, 48
  inspection, 48
  palpation, 49
    of the content, 52
      bimanual, 52
      single-hand, 52
      operations to carry it out, 50
      requirements inherent to the examinee, 49
      explorer requirements, 50
    of the continent, 50
      - inguinal rings, 51
      wall thickness, 51
      maneuvers in parietal hypertonia, 50
      Galambos method, 50 -
      palpation of the navel, 51
      parietal sensitivity, 51
      abdominal tension, 52
  percussion, 48
  written record of the examination,
  54 examination techniques, 46
Pathological abdomen, 573, 889
  auscultation, 591, 896
  inspection, 574, 889
    skin alterations, 574, 890 contour
    alterations, 574, 890 navel alterations, 574,
    890 volume alterations, 575, 890
    abnormal movements, 576, 892

  - palpation, 568, 892
    abdominal tension alterations, 578
      fluctuation, 578
      retinence, 578
      resistance, 578
        «belly on board>, 578
    of the continent, 892
    in meteorism, 578
    in pneumoperitoneum, 578
    in ascites, 578
  percussion, 573, 895
Accessory or spinal nerve, 214, 748
  anatomy and physiology, 214
  semidiagnosis, 748
  semiography, 748
  semiotics, 214

Acidosis, 1070, 1074
  definition, 1070, 1070 -
  metabolic, 1074, 1074
  respiratory, 1079, 1077
Achondroplasia, 338, 1131
Attitude(s), 13, 333, 713, 768, 875 -
  standing, 13, 336. 713
    in Parkinson's, 713
    in ataxia, 713
    in Korea, 714
    in hemiplegia 337, 713
    in spastic extension paraplegia, 714 in
    myopathies, 714
  in bed or bed, 333, 713
    shotgun trigger, 336, 714
    hemiplegic during coma, 714
  other pathological attitudes, 336, 713, 768, 875
  somatoconstitutional, 336
    asthenic type, 336
    aesthetic type, 336
  postural types, 14
Numbness and tingling, 55
Adrenoglomerulotropin, 1,052
Aerophagia or aerophagic belching, 853
Optical agnosia, 741
Water, regulation, 1 047
  neurohormonal control, 1,050
  income, 1,048
  losses, 1,048
Alkalosis. 1070, 1078
  definition, 1070
  metabolic, 1078
  respiratory, 1079
Aldosterone, chlorine and sodium regulation, 1 052
Albinism, 370
Amaurosis, 740
Amblyopia, 741
Amenorrhea, 1094
Anamnesis, 313
  -next, 314
    personal identity data, 315 history of
    current illness, 316
    reason for admission, 316
  remote, 317
    food, 317
    family pathological history, 317 personal
    pathological history, 317
    family tree, 317 lifestyle,
    317 toxic habits, 317
    Anisocoria, 450, 742
Anus, examination, 226,
918
  condylomas, 597, 918
  fistulas and abscesses, 597, 918
  fissures, 597, 918
  hemorrhoids, 597, 918
  prolapse, 597, 918
  exploration techniques, 226
  tumors, 918
Appendix, examination of,
  915 Blumberg maneuver, 915
  Rowsing maneuver, 915
  painful points, 915
    of Morris, 915
    of Monro, 915
    of Mc. Burney, 915
Spider veins, 372, 993 Anal
burning, 874
  gastric, 856
  lingual or buccolingual, 812
Joints and periarticular structures, 63, 401 physical
  examination, 63, 401
    cricoarytenoid, 408
    of the hip, 73, 413
    of the hand and fingers, 73, 411
    of doll, 72, 410
    knee, 75, 414 elbow,
    70, 409 shoulder,
    68, 408
      acromioclavicular, 69
      scapulovertebral, 69 foot,
    77, 418
    ankle, 76, 417
    sternoclavicular, 69
    sacroiliac, 68, 405
    temporomandibular,
Allergic arthritis, 425
  gouty, 425
  infectious, 423
  reactive, 424
  rheumatoid, 424
  septic, 423
Secondary arthropathies, 424
Arthroscopy, 409, 422
Ascites, 896, 969Ascites, liquid, 920, 971
  light yellow, 971
  cloudy yellow, 971
  biliary, 972
  thick, puriform, 972
  hemorrhagic, 972
  milky, 972
  mucinous, 972
  brown, 971
Asthenia, 1 114
Ataxia (s), 713, 728
  cerebellar, 729
  labyrinthine, 729
  medullary, 729
  mixed, 729
  peripheral, 729
  semitechnics, 158
  alterations in dynamic coordination, 729
  disturbances in static coordination, 729
Áomo, 1,042
  atom-gram, 1 043
  electron, 1,043

- structure, 1 042
- neutron, 1,043
- atomic weight, 1,043
- proton, 1,043
- Valencia. 1 043

Auscultation, 10
- definition, 10
- technique, 11
- cardiac, 126, 541
  - mitral opening click, 544
  - general considerations, 126
  - spotlights, 127
  - regions where it should be practiced, 127
  - noises, 128, 541
    - normal split, 130
    - pathological splitting, 543
      - of the first, 543
      - of the second, 544
    - rhythm modification, 543
      - bradycardia, 543
      - gallop rhythm, 545
      - fetal rhythm, 543
      - tachycardia, 543
    - modification in the timbre, 542
    - modification in intensity, 541
    - semiogenesis, 129, 543
      - of the first noise, 129, 543
      - of the second noise, 130, 544
    - third noise, 130, 545
  - Levine's crawling system, 128
  - systematic, 128
  - echo systoles, 546
  - murmurs, 130, 547
- of the kidney region, 1,025
- extracardiac vascular, 559

Auras, 772
- auditory, 772
- gustatory, 772
- motorboats, 772
- olfactory, 772
- sensitive, 772
- vegetative, 772
- visual, 772

Breast self-examination, 41
- testicular, 251

Armpits, physical examination,
- 37 palpation, 39

b

Bartel, hypoplastic status of, 355

Spleen, palpation,
- in dorsal recumbency. 235
- in Llanio position, 236 in
- Schuster position, 235 in
- Ziemmsen position, 235
- palpatory recognition, 236

Biotype or external habit, 13

Mouth, 216, 878
- labial commissures, 216, 878 -
  - commissural cheilitis, 879
    - cheilosis, 879
    - rágade, 879
- teeth, 217, 887
- gingiva or gums, 217, 888
- salivary glands, 220, 888
- lips, 216, 878
- tongue, 218, 880
- buccal mucosa, 217, 879
- hard and soft palate, 220, 888

Goiter, 1 121
- functional hypertrophy and hyperplasia, 122
- measurements, 1 122
- -palpation, 34, 1 122
  - Crile maneuver, 35
  - Lahey maneuver, 35
  - Marañón maneuver, 1 122
  - Quervain technique, 34
- inflammatory reactions, 1 122

Biopsies, neuromuscular, 759 -

pleural and pulmonary, 473
- renal, 1 028

Scrotal bags or scrotum, 245, 1,084
- magnification, 1,
- palpation of the vas deferens and cord

spermatic, 248, 1 084
- palpation of scrotum, 248, 1 084
- palpation of testicle and epididymis, 248, 1 084
- tunica vaginalis palpation, 248, 1 084

Borborygmos, 872

Brachycephalic, 25

Bradychory, 743

Bronchography, 475

Bronchoscopy, 473
- contraindications, 474
- indications, 474

Acute bronchitis, 477

Chronic bronchitis, 478

Bruxism, 815

Head scan 25
- face, 26
- skull, 25

Exertional cramp or intermittent claudication, 652
- of rest, 653

Calcium, 1,068, 1,187
- -hypercalcemia, 1069
- -hypocalcemia, 1,069

Cardiovascular system 100
- anatomy, 100 -

physiology, 109

Cardiac catheterization, 564

Cachexia, 339, 1 121
  Simmonds, 1 122, 1 154
Headache,
Cerebrospinal, fluid, 750
Blindness, color, 741
  psychic, 741
  verbal, 741
Cyanosis., 362
  hot, 364
  central, 362, 364
    circulatory, 363, 364
    pulmonary, 362, 364
  in vascular conditions, 658
  in slave, 364
  cold, 364
  mixed, 363
  peripheral, 362, 364
  by methemoglobin or sulfhemoglobin, 363
  toxic, 363,364
Collateral circulation, 373, 896
  arterial, 373
    coarctation of the aorta, 373
  venous, 373, 896
    lower cellar, 373
    upper cellar, 373
    portal, 374
    cup holder, 374
Intermittent claudication, 652
Clonus, 723
  of the kneecap, 723
  of the foot, 723Chlorine, 1 065
  hyperchloremia, 1,065
  hypochloremia, 1,065
Blood coagulation, 1 003
  factors, 1 004
Coccygodynea, 873
Endoscopic cholangiopancreatography, 943
Colon, examination of, 911,913, 914
Colonoscopy,948
Skin coloration, 20, 359 Vertebral
column, 64, 402
  cervical, 66. 403
  kyphosis, 65, 402
  dorsal, 67, 403
  scoliosis, 67, 402
  lordosis, 65, 402
  lumbosacral, 67, 403
  rectification, 67, 403, 404
  anatomophysiological account, 64
  semitechnics, 64. 402
Comma, 788
  carus, 790
  surpassed, 789
  vigil, 789
Body build, table of, 16
Communication, 2
  general aspects, 2 in
  medical practice, 2
  for physical examination, 2
Consciousness, level of, 146
Constipation, 861
  functional, 861
  organic, 861
Constitution, 344, 530
  acromegalic, 353
  Asthenic, Stiller, 355
  parcel inequalities of the, 354
  hypoplastic status of Bartel, 355
  thymolymphatic status of Paltauf, 355
  hypergenital, 354
  hyperthyroid, 353
  hypochromaffin, 354
  hypogenital, 354
  hypoparathyroid, 354
  hypothyroid, 353
  neuropathic, 355
  serological, 355
  sympathicotonic, 353
  vagotonic, 352
Constitutionology, 344
Seizures,771
Cretinism, 1 131
  endemic, 1 131
  sporadic, 1 131
Chronopathogram, 317
Neck examination, 33
  overall, 33
  the glasses of, 36
  rest of the structures, 36

CH

Chaumel gastric snap, 898 Mitral
opening snap, 544

d

Arterial dance, 537
Recumbent, 333
  active, 333
  passive, 333
Hippocratic fingers, 450, 530
Thoracic deformities, 454 localized
  or circumscribed, 454 vaulting, 454
    depressions, 454
  unilateral, 454
    dilation, 454
    retraction, 454
Thinness, 339, 1 120
Dermatome, 825
Dehydration, 1,057
  prior considerations, 1,057
  hypertonic, 1,058

- hypotonic, 1 061 -
mixed, 1 063
Diagnosis, 278
by comparison, 279
by exclusion, 279
by intuition, 279
by hypothesis or therapeutic, 279
by reasoning, 279
Diarrhea(s), 865
high,
868 low,
dysentery, 868
steatorrhea, 868
functional, 868
organic, 868
Diascopy, 357
Diastasis recti, 890
Diathesis, 355, 1 066
arthritic, 355
exudative, 355
fibrous, 355
hemorrhagic diseases of complex etiology, 1,006
due to platelet disorders, 1,006
due to vascular alterations, 1,006
due to disorders of plasmatic mechanisms or excessive circulating anticoagulants, 1,006 Teeth, 217, 887
of Hutchinson, 887
Digestive system, 216, 792
general anatomy and physiology, 792
oropharyngeal examination, 216
of the rectum and anus, 226
-general pathophysiology, 803
alterations in chewing, swallowing and salivary secretion., 804
mixed alterations, 804
motorboats, 803
secretory and absorption, 803
sensitive, 804
neoanatomy (physiological anatomy), 792
Dysphagia, 815, 818
functional, 818
-high location, 818
low location, 818
- medium location, 818
organic, 818
Dyspnea, 437
bradypnea, 437
continued, 517
de Biot, 439
recumbent, 515
by Cheyne Stokes, 438
of effort, 514
of Kussmaul, 439
cardiac, 511
expiratory, 437
general pathophysiology, 440
- inspiratory, 437
stridor goring, 437
drafting,
paroxysmal, 515
acute lung edema, 516 cardiac
pseudoasthma, 516
polypnea or tachypnea, 437
due to anoxia,
440 anemic,
440 anoxic,
440 histotoxic,
440 due to stasis,
440 general semidiagnosis, 442
Abdominal distension, 870
Dysuria, 1
023 Dolichocephalus,
Abdominal pain (ores), 825
epigastric, 833
non-epigastric, 835
quality or character, 842
agonizing or "excruciating", 842
constrictive, 843
cramp or torsion type, 842 flashing, 843
taxable, 842
lancinating, 842
pulsatile, 843
burning or burning, 842 dull,
843
terebrant, 842
of joint origin, 394
of muscle origin, 394
of bone origin, 394
of periarticular origin, 394
in gynecological conditions, 1,089
sharp, 1,089
chronic, 1,089
in kidney conditions, 1,014
nephritic colic, 1,014
simple or capsular, 1,014
precordial, 505
precordial pain, 509
anginal pain, 506
angor pectoris, 508
myocardial infarction, 508
acute coronary insufficiency without iclusion, 508
simple precordial pain, 509
rectal, 872, 873
proctalgia fugax, 873
theories, 827
of the vagal transmission of the reflex, 829
of pure visceral pain (Leriche), 827
of the intimate mechanism of the pain reflex, 829
of the peritoneosensitive reflex (Morley), 827
of the viscerosensory reflex (RossMackenzie), 827
eclectic pathogenic, 829
thoracic, 434
pleuroparietal and chest wall, 435 pulmonary
pleurovisceral, 435

sharp side point, 435
subacute side tip, 436 visceromotor
reflex, 435 viscerosensory
reflex, 435 semiogenesis, 435

peripheral vascular, 653
Duodenoscopy, 942

Echocardiography, 567
Echoencephalography, 754
Ectasia, 276
Age, pathology according to, 311
Edema (s), 377, 1 025
secondary aldosteronism, 378
whites, 381
soft, 382
tanned, 381
cardiac, 379
cyanotic, 381
cavitaries, 383
ascites, 383
hydropericardium, 383
hydrothorax, 383
saucepan or godet, 382
in liver cirrhosis, 380
widespread, 383
located, 380
nutritional, 380
kidney, 380, 1 025
diffuse glomerulonephritis, 380
nephritic syndrome, 380
registration by degrees,
383 ruddy, 381
semiogenesis, 377 -
visceral, 383
Electrocardiography, 568
fundamental electrocardiographic alterations, 583
atrial enlargements or hypertrophies, 583
rhythm disturbances (arrhythmias), 600
normotopic, 601
sinus arrhythmia, 602
sinus bradycardia, 602
migratory pacemaker, 603
sinus arrest, 603
sinus tachycardia, 601
heterotopic, 603
extrasystoles, 605
headphones, 605
nodes, 605
ventricular, 605
atrial fibrillation, 607
earphone flutter, 608
paroxysmal supraventricular tachycardia, 607
paroxysmal ventricular tachycardia, 607
due to driving disorder 609
atrioventricular blocks, 609
bundle branch blocks, 584
concept, 584
right branch, 585
left branch, 586
hemiblocks or fascicular blocks, 587 Wolff-
Parkinson-White syndrome, 610 ischemic
heart disease, concept, 593 permanent
acute coronary insufficiency
(heart attack), 594
evolutionary diagnosis, 594
positive diagnosis, 594
topographic diagnosis, 594
transient acute coronary insufficiency, 593 chronic coronary
insufficiency, 600
Subendocardial ischemia, 594
stress test (Master's test), 594
acute cor pulmonale, 614
chronic cor pulmonale, 614
pericarditis and pericardial effusions, 612
produced by digital, 614
produced by potassium, 614
ventricular overloads and growths, 588, 590
right, 589, 590
Cornell index, 588
Sokolow index, 588
White-Bock index, 588 left, 590
leads 570
electrocardiograph,
571 P wave study, 308 T
wave study, 574 U wave
study, 574 QRS complex
study, 574 PR interval study,
572 QT interval study, 574 PQ (or
PR) segment study, 572 ST
segment study, 574 fundamentals
electrophysiological, 568 method of reading
an ECG, 574 determination of the
electrical axis, 575 determination of the
rhythm, 575 determination of the
frequency, 575 electrical position, 580

Electroencephalography, 756
Electrolytes, 1,042
normal figures, 1 050, 1 053, 1 058
regulation of, 1 052
Electromyography, 758
Pregnancy (gestation), 1,098
ectopic, 1 102
Emprostóonos, 335, 714
Dwarfism, 338, 1 131
achondroplasia, 1 131
pituitary (infantilism), 1 133
thyroid, I 131
Gums, 217, 888
Endometriosis, 1,102

Addison's disease, 369, 1 221
  de Graves-Basedow, 1124
  by Mikulicz, 811
  by Nicolas and Favre, 918
  by Takayasu, 523
  by Vaquez-Osler, 361
  of Von Recklinghausen, 369 of
  Von Willebrand, 1 008
Subcutaneous emphysema,
  384 mediasthenic,
384 Enterorrhagia,
Medical interview, 309
Epistaxis, 851, 1,008
Balance, 1,047
  acidbase, 1 069
  electrolytic, 1,052
  water, 1,047
Palmar erythema, 878
Erythromelalgia, 686
Non-aerophobic belching, 855
Sclerotoma, 825
Scotomas, 741
Esophagus, 815, 888
  anatomy, 815
  physiology, 816
  histology, 816
Esophagoscopy, 930
Speculum, examination with it, 264
Spinal or accessory nerve, 214, 748
  anatomy and physiology, 214
  semidiagnosis, 748
  semiography, 748
  semiotics, 214
Sputum, 471
 Station, see Attitude
Marital status, 316
Mental state, 145
Statoacoustic, nerve, 204, 746
  -cochlear, 204, 746
    anatomy and physiology, 204
    - hearing loss,
    746 paracusis,
    746 Rinne test, 207
    Weber test, 206
    Schwabach test, 207
    semidiagnosis, 746
    semiography,
    semitechnics, 206
    tinnitus, 746
  vestibular, 207, 746
    anatomy and physiology, 207
    Babinsky star, 209
    Romberg maneuver, 209
    nystagmus,
      Bárány caloric test, 210 Bárány
      index test, 209 semiogenesis, 746

      of balance disturbances, 746
      of nystagmus, 746 -
      of vertigo, 746
    semitechnics, 209
Bartel hypoplastic status, 355
Paltauf thymolymphatic status, 355 Rales, 464

  cavernous, 467
  crackles, 465
  hoarse, 464
  wheezing, 465
  subcrackles, 466
Stethoscopes, 10
  binaural, 10
Stomach, 897
  anatomy, histology and physiology, 897
  probing examination, 952
    gastric aquilia, 953
    gastric juice, 952
  gastrosucorrhea,
  898 hyperchlorhydria,
  Hollander test. 954
  Kay test, 954Stomatitis, 879 aphthous,
  879
  catarrhal 879
  in hemopathies, 879
  eruptive, 879
  necrotic, 879
  nutritional, 879
  due to yeast (thrush), 879 toxic,
  880Stomarrhagia, 879
Imaging studies
  of the heart, 566
    angiocardiography, 566
    coronary angiography,
    566 Doppler echocardiography, 567
    traditional echocardiography, 567
    stress echocardiography, 567
    isotope perfusion studies, 567
    radiography in oblique position, 566
    fluoroscopy, 566
    nuclear magnetic resonance, 567 -
    teleradiography, 566
    computed axial tomography, 567
  of the nervous system, 752
    angiography, 753
    echoencephalography, 754
    myelography, 754
    pneumoencephalogram, 753
    -simple x-ray, 753
    nuclear magnetic resonance., 755
    computed axial tomography, 754
    photon emission tomography (SPECT), 756
    positron emission tomography (PET), 756
  of the respiratory system, 474
    angiopneumography, 475
    image-guided percutaneous biopsy, 476
    bronchography,
    475 echocardiogram,

    - scintigraphy, 476
    - simple x-ray, 475
    - fluoroscopy or fluoroscopy, 474
    - magnetic resonance imaging, 475
    - computed axial tomography, 475 simple
    - or conventional tomography, 475
    - ultrasonography, 476
  - of the urinary system, 1,027
    - aortography, 1 028
    - cavography, 1 028
    - cystography, 1 027
    - kidney scan, 1 028
    - ascending pyelography, 1 027
    - plain radiograph, 1 027
    - retropneumoperitoneum, 1 028
    - nuclear magnetic resonance, 1 028
    - computed axial tomography, 1 028
    - abdominal ultrasound, 1 027
    - excretory urography, 1 027
  - of the peripheral vascular system, 673
    - angiographies, 673
      - aortography, 673
      - phlebography, 673
      - lymphography,
    - 673 simple x-ray, 672
- Medical ethics, 4
- Etiology, 281
  - biological agents, 289
  - physical, 284
    - cold, 285
    - atmospheric pressure, 285
    - radiation, 285
  - genetics, 290
  - iatrogenic, 308
  - immunological, 301
  - mechanics, 284
  - neoplasms, 304
  - psychics, 308
  - chemicals, 286
    - alcoholism, 287
    - drugs, 287
    - air pollution, 288
    - non-smoking, 288
  - socioeconomic, 282
  - neoplasms, 304
- Eumetry, 152
- Exam
  - physical as part of a clinical record, 1 general, 12, 332
    - exploration techniques 12
    - model of your record, 24
    - pathological, general inspection, 332
  - regional, 25
  - by catheterization of the duodenum and bile ducts, 952
    - microscopic alterations, 954
    - bile A. 954
    - bile B. 954
    - bile C,954
    - biliary drainage, 954
    - timed drain, 957
    - elimination of bromosulfthalene, 957
- Exophthalmia, 1 124
  - bilateral, 1 124
  - unilateral, 1 129
- Thoracic expansion, 94
  - modifications of the, 456
    - bilateral, 456
    - located, 456
    - unilateral, 456
- Expectoration, 446
  - quantity, 446
  - color and transparency, 446
  - mucopurulent, 446
  - mucosa, 446
  - smell, 446
  - purulent, 446
  - flavor, 446
  - bloody, 447
  - serous, 447
  - viscosity and consistency, 446
- Expulsion of gases through the anus, 872

- Facial, nerve, 202, 744
  - anatomy and physiology, 202
  - ageusia, 745
  - hypogeusia, 745
  - parageusia, 745
  - central facial palsy, 744
  - peripheral facial palsy, 744
  - examination technique, 203
    - motor function, 203
    - sensory function, 204
    - Pierre-Marie-Foix maneuver, 204
- Facies, 14, 339, 711
  - acromegalic, 342
  - adenoid, 339
  - aortic, 341
  - cretinoid, 342
  - of asystole, 341
  - of capsular hemiplegia during coma, 711
  - of myasthenia gravis 712
  - of facial paralysis, 343, 711
  - by Uffenheimer, 1 197
  - of pseudobulbar, 711
  - painful, 343
  - febrile, 344
  - hepatic, 341
  - hyperthyroid, 342
  - mediastinal, 343
  - myxedema, 342
  - pneumonic, 342
  - parkinsonian, 343, 712
  - peritoneal, 341

  - kidney, 341
  - tetanic, 344, 712
  - typhoid or stuporous, 344

Fatigue, 652

Karolyi phenomenon, 815
  - by Nicoladoni-Bramhan, 657
  - by Raynaud, 684
  - de Vernet, 212

Phenotype, 291, 345

Uterine fibroid, 1 100

Fever (hyperthermia), 385
  - thermoregulatory centers, 386
  - febrile cycle, 388
  - thermogenesis, 386
  - thermolysis,
  - 386 febrile type(s), 387
    - continuous fever, 387
    - hectic fever, 387
    - intermittent, 387
    - inverted, 387
    - undulating, 388
    - recurrent, 388
    - sender, 387
  - semiogenesis or pathophysiology, 385

Arteriovenous fistulas, 682

Phlebitis, 662

Flictenas, 660

Rectal discharge, 874

Phonocardiography, 566

Phosphorus, metabolism, 1 056, 1187

Bronchial thrills, 458

Coldness of limbs, 662

Pericardial rub, 558, 643

g

Brain scan, 756

Cloquet's lymph node(s), 250
  - of the head and neck, 35
  - de Troisier-Virchow, 999

Focal gangrene, 660
  - wet, 660 dry,
  - 660

Cardiac output, 117, 563

Gastrochymogram, 952
  - Kay test, 954
  - Hollander test, 954

Gastroscopy, 935

Genotype, 291, 345

Hump, 402

Gigantism, 338, 1 133
  - brain (Sotos syndrome), 1 133
  - pituitary, 1 133
  - hypogonadal, 1 133
  - -local,1133

Gynecomastia, 1 240

Glossodynia or glossalgia, 812

Glossopharyngeal, nerve, 211, 747
  - anatomy and physiology,
  - 211 general considerations, 210
  - semidiagnosis, 747
  - semiography, 747
  - examination techniques, 212
    - taste examination, 212 Vernet
    - phenomenon, 212 carotid
    - sinus reflex, 212 pharyngeal reflex,
    - 212

Taste, alterations of, 813
  - ageusia, 813
  - cacogeusia, 813
  - dysgeusia, 813
  - sour flavor, 813
  - amnargo flavor, 813
  - sweetish flavor, 813
  - metallic taste, 813
  - pasty taste, 813

h

Chvostek's cirrhoic habit, 876

External habit or biotype, 13

Halitosis, 812

Painful hunger or gastralgochenosis, 840

Hematemesis, 851, 982

Hematopoiesis, 986

Hematuria, 1 018

Hemeralopia, 741

Hemianopsia, 740

Hemiplegia, 724
  - spastic, 726
  - flaccid, 725
  - larvae, 726
  - Barré maneuver (of the fingers), 726
  - topography, 726, 777
    - alternate, 777
      - bulbar, 778
      - peduncular, 777
      - protuberant, 778
    - capsular, 726
    - cortical, 726
    - subcortical, 727
    - thalamic, 727

Hemophilias, 1 006

Hemogasometry, 1,071

Hemolymphopoietic system, 233, 986, 995
  - anatomy and physiology, 233
  - hematopoiesis and main symptoms and signs, 986
  - main syndromes, 995

Hemoptysis, 447, 851
  - positive and differential diagnosis, 447
  - semidiagnosis, 448
  - circulatory conditions, 448
  - respiratory conditions, 448
  - hemorrhagic syndromes, 448

semiogenesis, 447
Haemostasis, 1 003
Hepatomegaly, 90S
  cirrhosis, 905
  hepatitis, 905
  cardiac liver, 905
  leukemic, 905
  parasitic, 909
  cysts, 905
  benign and malignant tumors, 905
Inheritance, 292
  autosomal dominant, 292
  autosomal recessive, 293
  X-linked dominant, 293
  mitochondrial, 295
  X-linked recessive, 294
  Y-linked recessive, 295 Hydrarthrosis,
424
Hydroelectrolytic, system, 1 042
Liver, 899
  atrophy, 911
  hepatomegaly, 905
  hepatoptosis, 905
  palpation of the 900
    Gilbert's bimanual, 900
    of Chauffard, 900
    from Devoto, 901
    of Glenard, 901
    by Mathieu, 901
    single-hand, 900
Hyperhydration, 1,064
Hiccups,
Pituitary, 1 137
  anatomy, 1 137
  physiology, 1 141
  histology, 1 139
Halibut major, 215, 748
  anatomy and physiology, 215
  semidiagnosis, 748
  semiography, 748
  semitechnics, 215
    tongue exam, 215
Arterial hypotension, 665
  orthostatic,
  666 primary,
  secondary, 666
Hypothermias, 388
  critical collapse, 389
Pupillary hippus, 198, 743
Clinical history definition, 1
  technique,
    in primary care, 311 in
    secondary care, 312 History of
current illness, 316 Antidiuretic hormone, 1,
050, 1,159
  production mechanism, 1 050
  osmoneurohormonal reflex, 1 050
  osmoneuropsychic reflex, 1 050
  tensiononeurohormonal reflex, 1 050
Gastrointestinal hormones, 960 Bones,
55, 397 shortening,
  398
  elongation, 399
  volume increase. 398
  crepitation, 399
  deformity, 397
  abnormal movements, 399
Skin moisture, 94, 371
  anhidrosis, 371
  bromidrosis, 371
  chromidrosis, 371
  hyperhidrosis, 371
  hypohidrosis, 371
  uridrosis, 372
    uremic frost, 372
Ilatrogenicity, 5
Jaundice(s), 366, 962
  due to increased formation of free bilirubin, 366, 64 due to
  disorders in the uptake of bilirubin in the hepatocyte, 366,
964
  due to disorders in conjugation, 366, 964
  due to disorders in excretion, 366, 966
  skin symptoms, 366
  circulatory symptoms, 366
  digestive symptoms, 366
    acholia, 366
    hypercolia, 366
    hypocolia, 366
  nervous symptoms, 366 tint,
  366
    flavinic, 366
    melanic, 366
    rubinic, 366
    verdinic, 366
Stroke, 277, 724
Functional impotence, 394, 725
  semiogenesis, 394
Body mass index, 351
    de Pignet, 351
    de Rohrer, 351
Infantilism or short stature, 1 133
  pituitary, 1 133
  primary ovarian, 1 133
Inspection, definition, 7
  technique of, 7
Interrogation, 317
    per system, 317
Radioactive isotopes in peripheral vascular conditions,
678

Laparoscopy, 923
  examinations performed by the, 929
  targeted biopsy, 929

-endoscopic surgery, 930
  laparoscopic cholangiography, 930
  splenoportography and manometry, 930
  laparophotography, 930
  cystography, 930
Language, alterations of the, 880
  syphilitic chancre, 887
  peeled, 881
  in skin conditions, 882
    of the nervous system, 887
    endocrine, 882
    hematological, 882
    systemic, 887
  fissured, scrotal or cerebriform, 882
  geographical, 882
  middle rhombic glossitis, 882
  leukoplakia, 881
  macroglossia, 881
  magenta, 882
  microglossia, 880
  downy black, 882
  dangerous, 882
  coated, 881
  dry, 882
Language, 149
  definition of terms, 149
  exploration, 150
  anatomophysiological significance, 150
Leucorrhoea, 1,096
Law of Distance of Irritating Thorns, 808 Von Wahl, 892

Congenital generalized lipodystrophy, 1 133
Lipothymia, 521

I

Gastric fullness or fullness, 857

M

Moms, 37, 1 098
  self-examination of the, 41
  physical examination of, 37
  -semiology, 1 098
Barré maneuver, 163
    separation of the fingers of the hands, 726
  Bragard's, 404
  de Brudzinski, 166
  skull compression, 66
  from Crile, 35
  de Déjerine, 404
  by Erischen, 406
  de Finck, 403
  of Glenard, 238
  Gordon's, 173
  by Jendrassik, 171
  de Kernig, 165
  from Marañón, 1,122
  by Mingazzini, 163
    of lower limbs, 163
    of upper limbs, 164
  deLahey, 35
  of Lasègue, 404
  of Leven, 899
  - from Lewin, 406
  de Menell, 407
  by Nafziger-Jones, 404
  - from Neri I, 404
  - from Neri II, 404
  of Oppenheim, 173
  fistpercussion, 240
  de Schaeffer, 173
  - from Tarral, 896
  -de Volkmann, 406
  FABERE, 406
Magnesium, 1 068, 1 189
  hypermagnesemia, 1
  068 - hypomagnesemia,
1 068 Achromic spots,
371 Colonic manometry,
958 - esophageal,
958 Walking, 12, 337, 396,
  715 small steps, 716
  ataxic, 337, 715
    cerebellar, 338, 716
    tabetic, 337, 7l6
- from "clawn", 717 -
from the side, 397
- from «duck» 397
  of "toad", 717 of
  Trendelenburg, 397 of
  the quadriceps, 397
  of the gluteus maximus;
  397 digitigrade, 338, 716
  in primary myopathies, 716 in
  scissors, 338, 716
  spastic, 715
  scything or hemiplegic, 337
- backwards, 397
- raising the pelvis, 397 paretic,
  715
  polineuritica or steppe, 337 "
  greeting", 397
Megakaryopoiesis, 992
Melanoderma, 369, 1 221
  diffuse, 369
  Addison's disease, 369, 1 221
  hemochromatosis, 369
  neurofibromatosis, 369 -
  localized, 369
  chloasma, 369
  ephelides or freckles,
  369 moles or nevi, 369
Mane, 860, 982

Memory, 149
Menstruation, disorders, 1 093 093
  amenOrrea,
  hypermenorrhea, 1 093
  hyperpolymenorrhea, 1 093 –
hypoligomenorrhea, 1 093
  hypomenorrhea, 1 093
  metrorrhagia, 1 093
  oligomenorrhea, 1 093
  opsoligomenorrhea, 1 093
  opsomenorrhea, 1 093
  polymenorrhea, 1 093
  proliomenorrhea, 1,093
Head metamer, 899
Meteorism, 870
Galambos method, 50
Overflow urination, 1,023
Mydriasis, 743
Migraine or migraine, 775
Miosis, 746
Myotome, 825
Myxoedema, 384, 1 178, 1 179
Molecule, 1 043
  molecule-gram, 1 043
  molecular weight, 1 043
Monoplegia, 724
Motility, 160, 724
  anatomy and physiology, 161
  general considerations, 160
  paralysis and paresis, 724
    due to central motor neuron injury., 724
    due to peripheral motor neuron injury, 724
  semitechnics, 160
    segmental muscle strength, 162
    Barré maneuver (lower limbs), 163
    Mingazzini maneuver, 163
    voluntary active motility, 162
Reason for admission, 316
Common oculomotor nerve, 194, 741
  semiography and special semidiagnosis, 742 intrinsic
  portion, 198, 743
    accommodation reflex, 198, 743
    convergence reflex, 198, 743
    consensual reflex, 198, 743
    photomotor reflex, 198, 743
  ophthalmoplegia,
    infranuclear, 742
    nuclear, 742
    supranuclear, 742
External oculomotor nerve, 194, 741 Abnormal
movements, 764
  convulsions, 769
  choreic, 767
    Sydenham's, 767
    Huntington's, 767
  dystonokinesias, 767
    torsion dystonia, 768
    spasmodic torticollis, 767
  muscle spasms, 768 -facial,
    768
    hiccups, 768
  fasciculations and fibrillations, 769
  hemiballism, 768
  psychogenic hyperkinesias, 769
  myoclonus, 766
  tremors, 765
    resting, 765
    intentional, 766
    permanent, 766 tics,
  768
Jugal mucosa, 223, 879
Vesicular murmur, 460
  intensity changes, 460
  of rhythm, 461
  of tone, 461
Muscle, 63, 399
  physical examination, 63, 399
  volume increase, 400
  volume decrease, 400

N

Nose, examination of the, 28
Nausea, 857
  Nephromegaly, 1,025
Pneumogastric, nerve, 210, 747
  anatomy and physiology, 212
  aphonia, 747
  general considerations, 210
  examination techniques, 213
  vocal cords, 213, 214
  carotid sinus reflex, 213 soft
  palate reflex, 213 pharyngeal
  reflex, 213
  oculocardiac reflex, 213
  semidiagnosis, 747
  semiography,
  747 bitonal voice,
Neuralgia(s), 436
  of the glossopharyngeal,
  747 of the trigeminal,
  775 phrenic,
  436 intercostal, 436
Neuroendocrine, regulation, 1 109
  role of the hypothalamus in, 1 109
  hormones, 1 109
  retrocontrol of hormones, 1 110
Cranial nerves, general considerations, 181
Nyctalopia, 741
Nocturia, 1,023
Nystagmus, 210, 746, 747
  eyepiece, 747
  central nervous system, 747
  vestibular, 210, 747
Phlebostatic level, 537

Osler's nodes, 527
rheumatic, 527
vascular, 661
Pathological nomenclature, definition, 275

Obesity, 338, 1 116
endogenous factors, 1 116
exogenous factors, 1 116
Obnubulation, 789
Odynia, 276
Odynophagia, 820
External ear, examination, 30
otoscope examination, 31
tympanic membrane, 32
Eye, exploration of external structures of the, 26
guidance for examination and registration, 26
- exam technique, 27
Olfactory, nerve, 185, 739
anatomy, 185
semiography, 739
olfactory hallucinations, 739
anosmia, 739
hyposmia, 739
parosmia, 739
exploration technique, 185
Oligoanuria, 1 020, 1 029
-postrenal, 1 022
prerenal, 1 020 -
renal, 1 020
Tarral transabdominal wave, 896
Opisthotonos, 335, 714
Opsyuria, 1,023
Optical, nerve, 187, 740
anatomy and physiology, 187
semitechnics, 190, 740
visual acuity, 190, 740
campimetry, 191, 740
quadrantanopsia, 741
hemianopsia, 740
heteronymous, 740
homonymous, 740
fundus, 191, 741
perimetry, 191,740
consensual reflex, 190, 198, 743
photomotor reflex, 190, 198, 743
color vision, 191, 740 Orientation,
147
Urine, exam, 1 023
density alterations, 1,024
of color, 1,023
deep yellow, 1 024
milky, 1 024
reddish brown, 1,024
brownish, 1 024
reddish or red, 1 024
- purplish red, 1 024
Addis count, 1 027
incontinence, 1 023
Oropharynx, 220, 888
white sore throat, 888
red sore throat, 888
Orthopnea, 333
Orthotones, 714
Hypertrophic pneumatic osteoarthropathy, 451
Osteogenesis imperfecta, 395
Osteomioarticular, system, 55, 392
anamnesis, 392
anatomy and physiology, 55
physical examination, 61
alterations of, 396
Ovary, volume increase, 1 102 solid
tumors, 1 105
Oximetry, 564

Q

Paleness,360, 655, 993
alabastrine, 360
waxy, 360
concept and semiography, 655
constitutional, 360
emotional, 361
-located, 360, 658
straw, 360
semiogenesis and semidiagnosis, 655
earthy, 360
greenish, 360
Palpation, definition, 8
technique, 8
Heart palpitations, 517
isolated, 517
in salvo, 518
atrial fibrillation, 520
earphone flutter, 520
paroxysmal tachycardia, 520
vascula;, 520
Paltauf, thymolymphatic status of, 355
Pancreas, 900, 957
physical examination, 900
palpation, 902
Grott method, 903
- Mallet-Guy method, 903, 911
Desdardins pancreatic point, 903
functional examination, 957
duodenal catheterization,
952 Panendoscopy,
943 Damoisseau parable, 488
Abdominal paracentesis, 919
Paraplegia, 714, 760
Paratype, 345
Parathyroid, 1 186
anatomy, 1 186

  physiology, 1 186
  histology, 1 186
Pathetic, nerve, 194, 741
Pathogenesis, 281
Hairs, 21, 1 134
  -examination of. 21
    guide to the examination of, 23
  hirsutism, 1 134
    category of hairs, 1 134
  primary trichogenesis, 22
  secondary trichogenesis, 22
Penis, 245, 248, 1 084
  exploration techniques, 248
  ulcerations, 1,084
Percussion
  - cardiac, 125, 538
    absolute dullness, 125, 538
    relative dullness, 125, 538
  definition, 8
  - of the thorax, 96, 458
    of pleural effusion, 458
  hypersonority, 458
  hyposonority, 458
  physiological and topographical modifications, 97
  techniques of the, 97
Weight, 15
  from 6 months to 20 years,
  15 men, table of, 16
  women, table of, 16
Reversal peristalsis, 813
Esophageal pHmetry, 958
Foot, 77,418
  calcaneus, 418
  dig, 418
  equino varus, 418
  hallux valgus, 418
  plane, 418
  valgus, 418
  Varus, 418
Skin, 19, 356, 381
  cracked or craquelé, 381
  exam, 19
  guide to the exam, 20
  inspection of the, 20, 356
    color and pigmentation, 20
      color alterations of the, 358
    hygiene, 20
    injuries, 20, 356
      skin elementals, 356
      vascular skin, 372
  palpation of the, 21,
    humidity, 21, 371
    temperature, 21, 385
    texture and thickness, 21
    turgor and mobility, 21
  skin examination record, 21
  semitechnics, 19, 356
Pyoarthrosis, 424

Heartburn, 822
Colic heartburn, 872
Pituita (esophageal regurgitation), 823
Pleurototonos, 335, 714
Cystic pleurisies, 459
Pollakiuria, 1,022
Rheumatoid polyarthritis, 424
Polydipsia, 1,115
Polyphagia, 1 116
Polyuria(s), 1 020, 1 115
  hypotonic, 1 020
  osmotic, 1 020
Pachón position, 123
  of Mohammedan prayer, 334
Posture, see Attitude
Potassium, 1l 067
  hyperkalemia, 1,067
  hypokalemia, 1,067
Evoked potentials, 758 Praxia,
159, 730
Origin, 316
Proctalgia, 873
Rectal prolapse, 874, 918
Prognosis, 280
Clinical propaedeutics, concept, 273
Prostate, volume increase, 1 089
  palpation, 1 089
Rectal protrusion, 874, 918
Adams proof(s), 675
  of Allen., 672
  of Carbamazepine, 1,161
- from thirst, 1 l61
  Miller, 1161
  by Oschner and Mahhorner, 676
  of Perthes, 678
  of Pratt, 676
  by Queckenstedt-Stookey, 751
  response to Pitresín, 1 161
  de Schober, 404
  for the study of muscular circulation, 670
  for the study of capillaries and small arteries, 670
  for the study of the venous system, 675
  for the study of vasomotor tone, 672
  for the study of the great arteries, 671
Anal itching, 874
Ptyalism, 808
Bids, 873
Arterial pulse, study, 133, 663
  amplitude alterations, 665
    alternating pulse, 665
    bigeminated pulse, 665
    filiform pulse, 665
    jumping pulse, 665
    rate disturbances, 136 rhythm
  disturbances, 664 complete
    arrhythmia, 665
    extrasystolic, 664
    intermittent, 664

- alterations of the arterial wall, 663
  alterations of synchronism, 663
  in peripheral vascular conditions, 663 axillary, 134

  ulnar, 134
  femoral, 135
  order, 135, 663
  popliteus, 135
  radial, 135, 663
  tibialis posterior, 135
Lumbar puncture, 749
  cerebrospinal fluid, 749, 750, 752
  technique,
  750 pericardial,
  565 site,
  technique, 565
Vaquez posterior auricular point(s), 533
  - from Erb, 555
  renoureteral pain, 239
  remote, 240
  earlier, 239
  later, 239
Purples, 372, 993, 1 003
  ecchymosis, 372, 993
  hematomas, 372, 993
  petechiae, 372, 993
  vibice, 372, 993

Queckenstedt-Stookey test, 751 Ovarian cysts, 1 105, 1 108 Cystography (cystography), 929, 930

R

Race, 316
  Straight, exam, 226,918
  tone alterations, 918
  foreign bodies, 918
  stenosis, 918
  fecalomas, 918
  crouching position, 229
  genupectoral position, 229
  gynecological, lithotomy or dorsosacral position, 229
  standing position, 229
  Sims position, 229 rectal
  examination, 230, 918
  tumors, 918
Rectal bleeding,
873 Rectosigmoidoscopy, 944
Reflectivity, 166, 720
  superficial areflexia, 722
  examination of reflexes, 166
  hyperreflexia, 722
  hyporeflexia and areflexia, 721
  semiography and semidiagnosis, 720
Reflex (s), 167, 720
  cutaneous-mucosal (superficial), 171
    corneal or conjunctival, 171
    cremasteric, 172
    cutaneous-abdominal, 172
    cutaneous-plantar, 172, 722
    pharyngeal,
  osteotendinous or deep, 167 achilleus,
    171
    bicipital, 168
    ulnapronator, 169
    of the finger flexors, 169
    of the orbicularis, 168
    of the long supinator, 169
    masseteric, 168
    midpubic, 170
    patellar, 170
    tricipital and olecranon, 169
Hepatojugular reflux, 649
Cervical region, 33, 66, 403, 536
  arterial beats, 536
  arterial dance, 537
  venous repletion or engorgement, 537
  Cardarelli-Oliver sign, 537
  venous beats, 537
Precordial region, 120, 531
  tip shock, 122, 532, 533 beat, 122, 532

      in dome, 532
      epigastric, 532 in
      staircase, 532
      negative, 532
      universal, 532
    semiogenesis, 122
    situation, 123
  inspection, 122, 531
  palpation, 123, 533
    clash at Bard's dome, 533
    valve shocks, 533
    pericardial rub, 535
    technique of, 123
    thill, 534, 662
      continuous, 535
      diastolic, 535
      systodiastolic, 535
      core systolic, 535
        mesocardial, 535
      vascular, 662
Regulation, neuroendocrine, 1 109
Gastric regurgitation, 855
Player, system, 241, 1 081
  female, 252, 1,089
    anatomy and physiology, 252, 256
    physical examination, 261
    semiology and pathological processes, 1 089
  male, 241, 1,081
    testicular self-examination, 251

anatomy, histology and physiology, 241 physical
examination, 246
semiology and pathological processes, 1 081
Breathing, by Biot, 437, 439
Cheyne-Stokes, 437, 438
Kussmaul, 437, 439
Raciborsky's breathy, 461
Respiratory, system, 80,431
clinical anatomy, 80
anamnestic data, 431
clinical physiology, 86
main symptoms, 433
Infectious rheumatism, 425
acute polyarticular, 425
Kidney, 237, 1 009
biopsies, 1,028
ultrasound, 1 027
endoscopic examinations, 1,027
imaging examinations, 1,027
aortography, 1,028
cavographies 1 028
cystography and urethrography, 1,027
nuclear magnetic resonance, 1,028
retropneumoperitoneum, 1,028
simple, 1,027
computed axial tomography, 1 028
ascending urography (pyelography), 1 027
descending, 1 027
scintigraphy, 1028
palpation, 237
Guyón maneuver, 237
Glenard maneuver, 238
Goelet method, 239 -
renal rally, 238
functional tests, 1 027
Gallop rhythm, 545
diastolic, 546
earphone, 546
ventricular, 546
semiogenesis,546
systolic, 546
Pleural rubbing or rubbing, 457, 463
Rubicundes, 361, 658
congenital, 361
- emotional, 361
in vascular conditions, 658 toxic,
361Venous rumor, 558
in portal hypertension, 558

Yes

Low back pain, 425
acute, 427
common vertebral sciatica, 428
chronic, 428
Epigastric jump, 535, 858
Health, 281
Semiodiagnosis, definition, 274, 275
Semiogenesis or pathophysiology, definition, 274, 275
Semiography, definition, 274, 275
Semiology, definition, 273
Semiotechnics, definition, 274, 275
Paranasal sinuses, examination of the, 28
Sensitivity, 174, 734
exploration of the, 174
barsesthesia, 179
barognosia, 179
bathyesthesia, 179
painful, 178
stereognosia, 179
palesthesia, 179
tactile, 178
thermal, 178
visceral, 178
semiography and semidiagnosis, 734
Pseudojaundice, 368
carotinic, 368
picric, 369
Sex, 315
Sialodochitis, 809
Sialophagia, 808
Sialorrhea, 808
Sign(s), definition, 276
Duroziez cardiovascular, 559
of Ewart or of Pins, 643
Mahler (ladder pulse), 657
de Roessle, 530
cushion, 334
by Cardarelli-Oliver, 537
digestive of Bouveret or Cruvelhier, 899
by Burni and Shuth, 557
of Cullen, 890
Courvoissier Terrier, 909
from Körte, 911
of Kussmaul, 892
of the Glenard strip, 899
by Roger, 808
of the iceberg, 894
Leven's sign pain, 899
Argyl-Robertson nervous system, 743
of Babinsky, 172
substitutes, 173
Gordon maneuver, 173
of Oppenheim, 173
de Schaeffer, 173
of Brudzinski (contralateral), 166
of Brudzinski (nape), 166
by Charles Bell, 343, 745
of the pipe smoker, 711, 725
de Kernig, 165
of the gear wheel, 770
osteomyoarticular of the tip, 429
de Laségue, 429
heel, 428
from Trendelenburg, 413
of the Seze bell, 429

Chvostek parathyroid, 1 197
  by Escherich, 1 197
  by Frankl-Hochwart, 1 197
  by Hoffmann, 1 199
  by Kahn and Falta, 1 197
  - from Kashida, 1,199
  of Lust, 1 197
  of Pool, 1 199
  by Schlesinger, 1 197
  by Schultze, 1 199
  de Trousseau, 1 197
  de Weiss, 1 197
Bacelli's respiratory tract, 469
  de D'Espine, 469
  of the Pitres coin, 469
  of Skoda (scodism), 458
  of the Pitres string or plumb line, 454
Rogoff's adrenal gland, 1 218
Ballet thyroid, 1 177
  by Claude-Bernard-Horner, 1 177
  of Dalrymple, 1 177
  of Mobius, 1 177
  from Rosenbach, 1,177
  from Stellwag, 1 177
- by Von Graefe, 1,177
Homans peripheral vascular, 678
  de Neuhoff, 678
  of Rosenthal, 678
  of Unschuld, 678
Syncope, 521
  cardiac, 522
  postcardiac or cerebral, 522
  precardiac, 521
Cardiovascular
  syndrome(s), 278, 616
    Stokes-Adams, 610
    carotid sinus, 523 arterial
    hypertension, 627 venous
    hypertension, 648 arterial
    hypotension, 665 heart
    failure, 618 coronary
    insufficiency, 626
      angina pectoris, 626
      myocardial infarction, 626, 627
    pericardial, 641
      acute pericarditis, 341
      constrictive pericarditis, 644
    shock or shock, 616
    cardiac tamponade, 646
    valvular, 632
      aortic stenosis, 632, 638
      mitral stenosis, 632
      pulmonary stenosis, 632, 640
      tricuspid stenosis, 632, 635
      aortic insufficiency, 632, 638 mitral
      insufficiency, 632
      pulmonary insufficiency, 632, 641
      tricuspid insufficiency, 632, 637
      mitral valve prolapse, 632, 634 definition, 278

- acid-base balance, 1,074, 1,078
    metabolic acidosis, 1,074
    respiratory acidosis, 1,074
    metabolic alkalosis, 1,078
    respiratory alkalosis, 1,078
  of electrolyte balance, 1 064,
    default, 1,064
    for excess, 1,064
  of water balance, 1 057,
    hypertonic dehydration, 1058
      hypotonic, 1061
      mixed, 1063
    hyperhydration, 1,064
  digestive, 960
    ascites. 969
    carcinoid, 877
    de Koenig, 872
    by Peutz-Jeghers, 880
    Plummer-Vinson, 822, 882
    of Sjögren, 811
    Zollinger-Ellison, 954, 1 192 chronic
    diarrheal, 976 dysenteric,
    980
    tropical sprue, 978
    glossoenteroanemic, 812
    icteric, 962
    digestive bleeding, 982
    pseudoulcerous, 838
  endocrine gonadal, 1 237
    hypergonadism 1 246
      female, 1 246
        hyperstrinism, 1 246 -
        hyperluteism, 1 248
      male, hypergenitalism in men, 1 246
    hypogonadism, 1 237
      female, 1 243
        castration, 1 243
        hirsutism, 1 134
        intersexuality and hermaphroditism, 1,246
        menopause, 1,243
        Stein-Leventhal syndrome, 1,245
        Turner syndrome, 1,243
      male, 1,237
        male climacteric, 1 240
        by Klinefelter, 1,240
        sterility and impotence coeundi, 1 241
        eunuchoidism, 1 238
        adult geldings, 1,238
        prepubertal castrates, 1,238
        gynecomastia, 1,240
  pituitary, 1,148
    anterior lobe, 1 148
      hypertuitarism, 1 148
        acromegaly, 1 149
        gigantism, 1 151
      hypopituitarism, 1l 153

in adults (Sheehan), 1 154 in
childhood, 1 157
posterior lobe, 1 159
diabetes insipidus, 1 159
parathyroid, 1 190
primary hyperparathyroidism, 1 190
secondary hyperparathyroidism, 1 194
hypoparathyroidism, 1 194
tetany, 1 196
thyroid, 1 171
hyperthyroid, 1 172
hypothyroid, 1 178
congenital, 1 185
juvenile, 1 183
spontaneous myxedema of the adult, 384, 1 178, 1 179
adrenals, 1 207
cortical hyperfunction, 1 208
adrenogenital, 1 211
Cushing's, 1 208
of feminization, 1,213
of hyperaldosteronism, 1,215
cortical hypofunction, 1,218
acute (Waterhouse-Aderichsen syndrome), 1,218 chronic (
Addison disease), 1,221
- medullary hyperfunction (pheochromocytoma), 1 224
general(s) hyperglycemic, 1 229
hypoglycemic, 1 231
hypoglycemic coma, 1 235
pharmacological interruption of
gluconeogenesis, 1 232
due to defective production, 1 232
absolute, 1 232
relative, 1 233
due to excessive use, 1,232
tall size, 1 133
endocrine, 1 133
genetic, 1 133
hypothalamic, 1 133
nutritional, 1 133
other, 1 133
low size, 1 130
pituitary, 1 131
hypothyroid (dwarfism), 1 131
hemolymphopoietic, 995
adenic, 998
anemic, 995
splenomnegalic, 1,000
purpuricohemorrhagic, 1 003
neurological (s), 760
cerebellar, 779
comatose, 788
convulsive, 771
by Claude Bernard-Horner, 449
by Millar-Gübler, 778
of pyramidalism, 724
from Pourfour du Petit, 450
by Ramsay-Hunt, 745
de Weber, 777
extrapyramidal, 762
hemiplegic, 724
intracranial hypertension, 785
meningeal, 783
paraplegic, 760
parkinsonian, 769
sensitive, 734
headache, 774
dissociation of sensitivity, 734
peripheral, 735
syringomyelic, 734
tabetic, 734
spinal cord, 734
medullary conus, 737
anterolateral cords, 737
posterior cords, 737 Brown-Sequard, 724 paraplegic
sensory, 734 peripheral,
738 radicular,
737
ponytail, 738
brainstem, 775
bulbar, 778
anterior bulbar, 779
posterior bulbar, 779
of Avellis, 779
of Jackson, 779
Schmidt's, 779
Wallenberg's, 778
peduncular, 777
by Benedikt, 777
upper Foville, 777
Weber, 777
pontines, 778
from Lower Foville, 778
from Millard-Gübler, 778
osteomyoarticular, 423
compartmental, 429
sharp, 429
of the anterior tibial compartment, 430
of carpal canal tunneling, 410 joint
inflammation, 423
sacrolumbalgia, 425
paraneoplastic, 1,194
respiratory, 477
bronchial, 477
bronchiectatic, 480
acute bronchitic, 477
chronic bronchiotic, 478
bronchial asthma, 479
bronchial obstructive, 479
of adult respiratory distress, 499
pulmonary hyperventilation, 515
respiratory failure (IR), 494
combined, 497
diffusive,
497 in perfusion,
496 pathophysiology,

ventilatory, 496
obstructive, 496
restrictive, 496
mediastinal, or mediastinal, 492
pleural, 487
gaseous interposition, 491
liquid, 487
lungs, 482
atelectatic condensation, 485 -
inflammatory, 482
tumorous, 486
emphysematous, 482
urinals, 1,029
acute renal failure, 1,029 chronic renal
failure, 1,030
uremia, 1,033
nephrotic, 1,035
peripheral vascular, 679
acrocyanosis, 679, 685
arterial, 679, 681
aneurysms, 681
Raynaud's phenomenon, 679, 684
arteriovenous fistulas, 681, 682
peripheral arterial insufficiency, 681
acute arterial ischemia, 681
chronic ischemia, 681
erythromelalgia or erythemalgia, 679, 686
lymphatics, 679, 683
acute lymphangitis, 683, 684
lymphedema, 683
lymphatics, 679, 683
livedo reticularis, 679, 686
venous (s), 679
venous obstructions, 679, 680
thrombophlebitis and phlebothrombosis, 680
vaices, 679
Syndromology, 273
Symptoms, definition, 276
far away or at a distance, 277
Common. 277
of hormonal origin, 277
fluctuating, 277
generals, 277
turn signals, 278
premises, 277
objectives, 276, 277
pathognomonic, 277
prodromal, 277
reflections, 277
subjective, 276, 277
toxic, 277
Cardiovascular or circulatory system, 100, 120, 501
- digestive, 216, 792
metabolic endrocrine, 1 109
hemolymphopoietic, 233, 986
nervous, 145, 154, 181, 709
osteomyoarticular, 61, 392
reproductive, 241,252, 1 081
respiratory, 80, 91,431
urinary, 237, 1 009
peripheral vascular, 133, 650
Echo systoles, 546
Sodium, 1,066
hypernatremia, 1 066
hyponatremia, 1 066
Somatometry, 351
Somatoscopy, 352
Cardiovascular murmur(s), 130, 547 Leatham
classification, 55 1
general considerations, 130, 547
duration, 131, 549
intensity, 131, 547
irradiation, 131, 549
modifications experienced, 131, 550
highest intensity site(s), 131, 549
time or moment of cardiac revolution in
that occur, 131, 548
ringing, 131, 548
tone, 131. 548
diastolic, generalities, 555
of organic mitral stenosis, 555
of relative mitral stenosis, 556
of tricuspid stenosis, 556
of aortic insufficiency, 557
of pulmonary failure (Graham-Steell), 557 systolic, 131,
551
inorganic or accidental of the base, 131
inorganic or accidental of the tip, 131, 551
of ventricular septal defect, 554
of aortic stenosis, 553
of relative aortic stenosis, 554
of pulmonary stenosis, 552
of relative pulmonary stenosis, 553
of mitral incompetence, 551
of mitral insufficiency, 551
of tricuspid incompetence, 552
of tricuspid insufficiency, 552
of patent ductus arteriosus, 553
due to valve or chorda tendinea rupture, 555
pulmonary, 99, 462
amphoric, 463
cavernous, 462
glottic, 99
pleural,
462 tubal,
Stiller, asthenic constitution of, 355
Adrenals, 1 201
anatomy, 1 201
physiology, 1 204
histology, 1 201

Rectal examination, 230, 918
vaginal, 267, 1 098

Height, 6 months to 20 years, 15
  alterations, 1,129 -
    low, 1,130
      constitutional, 1 031
      endocrine, 1 130
      genetics, 1 133
      nutritional and metabolic, 1 031
      bone origin, 1 031
      others, 1,031
    and weight men table 16
  and weight, women, table, 16
Taxia, 154, 713
  dynamic coordination, 158
    finger-finger, 158
    -index-nose, 159
    heel-knee, 159
  static coordination, 158
    sensitized Romberg, 158
    Romberg sign. 158
Technique (s), from Chevassu, 249 -
  from Crile, 35
  de Quervain, 34
  de Lahey, 35
  by Osler, 537
  of the physical examination, 7
    auscultation, 10, 11
      immediate, 10
      mediate, 10
    inspection, 7
    palpation, 8
    percussion, 8
Temperament, 350
  cyclothymic, 350
  schizothymic, 350
Body temperature, measurement, 16
  -guide for axillary measurement, 18
  guide for oral intake, 19
  guide for straight shot, 18
Local temperature, 389, 662
Tendons, 57
Rectal tenesmno., 873
  bladder, 1 023
Tension, 52, 137, 627
  abdominal, 52
  arterial, 137, 627
    semiogenesis or production mechanism, 135
    semitechnics, 141
      direct measurement, 140
      auscultatory method, 141
      oscillometric method, 141
      palpatory method, 141
    physiological variations, 143
    individual variations, 143 regional
    variations, 144 venous, 648

Testicle, 241, 248, 1 084
  increase in size. 1 084
  exploration techniques, 248
Typology or phenotype, 346
  German classification, 349
  French classification, 348
  Italian classification, 346
  Constitutional type(s), 336 athletic,
  349
  asthenic, 13. 336
  asthenicoleptosomal, 349
  - breviline, 13, 347
  cerebral, 349
  digestive, 349
    dysplastic, 350
  sthenic, 13, 336
- longilinear, 13, 347
  muscular, 349
  normolineo, 13, 347
  picnic, 350
  respiratory, 93, 349
  of thorax, 452
  Lloyd Brown's Postural Positions, 14
Thyroid, 33. 1 165
  volume increase, 1 121
  anatomy, 1 165
  embryology, 1 165
  gland exploration, 33
  physiology, 1 166
  histology, 1 166
Muscle tone, 164, 723
  dystonia, 724
  hypertonia, 165, 723
  hypotonia, 165, 724
  semitechnics, 786
Thorax, 80, 91
  clinical anatomy, 80
  lines and demarcations, 91
  pathological, 452
    kyphoscoliotic, 453
    conoid or enlarged, 454
    shoemaker's, 453
    emphysematous, 452
    infundibuliform (pectus excavatus), 453
    normal, 92
    rickety, 453
    consumptive or paralytic, 452
  percussion, 96
Cough, 442
  consequences of, 445
  emetic, 445
  wet, 442
  intensity and frequency, 444
  rhythm, 445
    quintasa, 445
  dry, 445
  semiogenesis or pathophysiology, 442
  tonality and timbre, 445
    voiceless, 445
    bitonal, 445
    snore, 445

- Transillumination, 30
- Hutchinson's triad, 887
- Garland's triangle, 459
  - Grocco's, 459
- Trigeminal, nerve 199, 744
  - abolition of reflexes, 744
  - anatomy and physiology,
  - 199 dissociated anesthesia,
  - 744, 744 the, neuralgia of
  - paralysis of chewing muscles, 744
  - paresthesias, 744
  - exploration techniques, 202
  - trophic and secretory disorders, 744
  - lockjaw,344, 744
- Trophism, 116, 718
  - joint disorders, 719
  - skin and nail disorders, 375, 718
  - alterations of myotrophism, 719
    - muscular atrophies, 719
      - of spinal origin, 719
      - of muscular origin, 719
      - of peripheral nervous origin, 719 of
      - radicular origin, 719
    - pseudohypertrophies, 719
  - osteoporosis, 719
  - semitechnics, 166
- Thrombocythemia, 1,006
- Thrombophlebitis and phlebothrombosis, 679, 680
- Thrombocytopenias, 1,006
- Thrombocytosis, 1,006
- Tubes, increase in volume, 1 102

- Ulcers in lower limbs, 661 Units of
- measurement, 1,043
  - volume, 1,044
  - - milliequivalent, 1 044
  - millimole, 1044
  - milliosmol, 1 045
- Nails, 23, 375
  - exam, 23
  - deviation from normality, 24
  - exam guide, 24
  - example of registration, 24
  - pathological, 24, 375
    - flattened (platonychia), 24, 376
    - concave, spoon-shaped (koilonychia), 24, 376
    - Lindsay, 24, 375
    - Terry's, 24, 375
    - thickened (pachyonychia), 376
    - in watch glass, 24, 375
    - excavated, 24,376
    - frail (onychorrhexis), 376
    - curved, hooked (onychogryphosis), 376
    - Beau lines, 24, 375
    - Mees lines. 24, 375
    - Muehrcke line, 24, 375
    - gnaws (onychophagia), 24, 376
- Urethra, palpation, 250
- Urinary, system, 237, 1 009
  - anatomy and physiology, 1 009
  - examination techniques, 237
- Uterus, anatomy, 254
  - volume increase, 1 098, 1 099
  - exploration techniques, 263

- Vagina, anatomy, 254
  - exploration techniques, 263 Varicose
- veins, 679
- Peripheral vascular system, 650
  - anatomy and physiology,
- 650 Vectocardiography,
- 567 Circulatory velocity, tests, 563
  - arm to periphery, 563
  - arm to lung, 563
  - lung to periphery, 564
- Pratt's sentinel veins, 661
- Pulmonary ventilation, 88
- Vergeturas,
- Vertigo, 520, 746
- Gallbladder, palpation, 901
  - Abraham maneuver, 901, 902
  - Fiessinger maneuver, 901, 902
  - Murphy maneuver, 901, 902 Pron
  - maneuver, 902
  - vesiculomegaly, 909
- Vocal vibrations, 95, 456
  - preserved, 95, 457
  - pathological modifications, 457
    - abolition, 457
    - increase, 457
    - decrease, 457
  - origin, 95
  - semiogenesis, 456
  - transmission, 95
  - physiological variations, 95
- Navicular belly in skiff, 892 Pilar virilism
- or hirsutism, 1 134
  - clinical picture, 1 134
  - of central origin, 1,135
  - of hormonal origin, 1,135
  - of medicinal origin, 1,135
- Vitiligo, 370
- Volemia, 562
- Vomica, 448
  - bronchopulmonary, 448
  - extrathoracic or abdominal, 448
  - mediastinal, 448
  - pleural, 448
  - semidiagnosis, 448
  - semiography, 448
- Vomiting, 845
  - watery, 846

food, 846
bilious, 846
central, 846
foreign bodies, 846
fecaloid, 846
mucous, 846
peripheral, 846
porraceous, 846
bloody, 846
Voice, auscultation, 468
amphorophony or amphora voice, 468
bronchophony, 468
egophony, 468
normal, 468
pectoriloquy, 468
voiceless, 469
Vulva, anatomy, 254
exploration techniques, 263

x

Xanthomas, 876Xerostomia, 812

Traube area, 897 Labbé,
897
Mendel's pain, 898